A Dissertation on

SUBJECTIVE AND OBJECTIVE ANALYSIS OF URINARY BLADDER MORBIDITY FOLLOWING TYPE III RADICAL HYSTERECTOMY FOR CARCINOMA UTERINE CERVIX

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BONAFIDE CERTIFICATE

This is to certify that **Dr. P. Senthil Kumar, M.S.,** bonafide student of M.Ch. Surgical Oncology (August 2012 to August 2015) in the Department of Surgical Oncology, Government Royapettah Hospital, Kilpauk Medical College, Chennai has done this dissertation on "**Subjective and Objective Analysis of Urinary Bladder Morbidity following Type III Radical Hysterectomy for Carcinoma Uterine Cervix"** under the guidance and supervision of **Prof. R. Rajaraman, M. S., M. Ch.**, Head, Centre for Oncology, Professor, Head of the Department, Department of Surgical Oncology, Government Royapettah Hospital, Kilpauk Medical College, Chennai in partial fulfillment of the regulations laid down by The Tamil Nadu Dr. M. G. R. Medical University, Chennai for awarding **M. Ch. Surgical Oncology degree** in the Examination to be held in **August 2015**.

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DECLARATION

I, **Dr. P. Senthil Kumar**, **M. S.,** bonafide student of M.Ch. Surgical Oncology (August 2012 to August 2015) in the Department of Surgical Oncology, Government Royapettah Hospital, Kilpauk Medical College, Chennai solemnly declare that this dissertation on "**Subjective and Objective Analysis of Urinary Bladder Morbidity following Type III Radical Hysterectomy for Carcinoma Uterine Cervix"** was done by me at Government Royapettah Hospital, Kilpauk Medical College, Chennai between January 2013 to January 2015 under the guidance and supervision of **Prof. R. Rajaraman, M. S., M. Ch.**, Head, Centre for Oncology, Professor, Head of the Department, Department of Surgical Oncology, Government Royapettah Hospital, Kilpauk Medical College, Chennai.

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CONTENTS

INTRODUCTION

Voiding dysfunction after pelvic plexus injury occurs most commonly after abdominoperineal resection and radical hysterectomy. The true incidence of neurogenic vesicourethral dysfunction after various types of pelvic surgery is unknown, because there are few prospectively studied series of patients with preoperative and postoperative urodynamic evaluation.

The incidence has been estimated to range from 20 % to 68 % of patients after abdominoperineal resection, 16 % to 80 % after radical hysterectomy, 20 % to 25 % after anterior resection, and 10 % to 20 % after proctocolectomy [1]. These are estimates drawn from past literature and the current incidence is most likely significantly lower, owing to the use of nerve-sparing techniques during these types of pelvic surgery. A few studies have suggested that the bladder dysfunction after radical hysterectomy may be transient and that bladder function may recover to baseline within 6 to 12 months.

It has been estimated that in 15 % to 20 % of affected individuals, the voiding dysfunction is permanent [2]. The injury may occur as a consequent to denervation or neurologic decentralization, tethering of the nerves or encasement in scar, direct bladder or urethral trauma, or bladder de-vascularization. Neoadjuvant and/or adjuvant radiotherapy may play a role as well. The type of voiding dysfunction that occurs is dependent on the specific nerves involved, degree of injury and any pattern of re-innervation or altered innervation that results over time.

REVIEW OF LITERATURE

Neuroanatomy and Neurophysiology of Lower Urinary Tract

Precise knowledge of neuroanatomy and neurophysiology is important to understand to avoid urinary bladder morbidity during pelvic surgery as well as to treat lower urinary tract dysfunction.

Lower Urinary Tract

The lower urinary tract (urinary bladder and urethra) functions in a co-ordinated manner to bring about adequate bladder filling with perfect continence, and periodic complete voluntary emptying without any post-void residual urine and without reflux into ureter. It executes this function with appropriate sensation, between voluntary voiding up to a volume that is sufficient to prevent urinary frequency and without any pain or urgency. In the adult the lower urinary tract has voluntary and involuntary neural control, but it is normally under voluntary neural control, like defecation, which is clearly different from other visceral organs innervated by the autonomic nervous system whose regulation is solely by involuntary mechanisms [3].

The micturition process can be visualized as a complex of neural circuits in the brain and spinal cord that coordinate the activity of smooth muscle in the bladder and urethra [4]. These circuits act as on-off switches to alternate the lower urinary tract between two modes of operation: storage and elimination.

The urinary bladder can be divided into two parts: a body lying above the ureteral orifices and a base consisting of the trigone and bladder neck, because the two areas are

different but homogeneous within themselves regarding neuromorphology and neuropharmacology [5]. Histological examination of the bladder body reveals that myofibrils are arranged into fascicles in random directions [6].

The bladder outlet is composed of the bladder base, urethra, and external urethral sphincter. The bladder base has a laminar architecture with a superficial longitudinal layer lying beneath the trigone. A muscle layer deep to the superficial layer is continuous with the detrusor [7]. The smaller muscle bundles of the deep muscle layer in the bladder base exhibit a predominantly circular orientation which is the internal urethral sphincter [8].

In women, the urethra extends throughout the distal third of the anterior vaginal wall from the bladder neck to the meatus. The urethra is composed of tissues that aid continence rather than a single discrete and visible "sphincter." A network of vascular sub-epithelial tissue in women contributes to a urethral seal effect [4].

The bladder neck serves an important function in reproduction. In men, closure of the bladder neck facilitates antegrade ejaculation. This is accompanied through a rich noradrenergic innervation by sympathetic nerves that actively contract the bladder neck during ejaculation. However, in women, the density of adrenergic innervation in the bladder neck is reportedly less than that in men [4].

The relationship between bladder shape, size, pressure, and tension was expressed by the Laplace law [8]. Marquis Pierre-Simon de Laplace (1779 to 1827) has been called the Newton of France. Laplace made the keen observation that the tension in the wall of a

container necessary to contain a given pressure is directly proportional to the radius of curvature at any point. This is the Laplace law [8].

The Laplace equation states that there is a direct relationship between wall tension and intravesical pressure and bladder size. In this equation, T is tension, P is intravesical pressure, R is urinary bladder radius, and d is wall thickness. During bladder filling, P is relatively constant. With a fully distended bladder, d, because of its relative thinness, is ignored relative to the other parameters unless a hypertrophied wall exists. Thus, $T = P \times R/2$ approximates tension in the full normal bladder [8].

The urinary bladder performs several important functions. First, it must store an adequate volume of urine. The bladder wall must be able to stretch and rearrange itself to allow an increase in bladder volume without significant rise in pressure. In other words, the bladder wall must be highly compliant. Second, the smooth muscle and intrinsic nerves have to be protected from exposure to urine by the urothelium, which itself must also expand readily during filling. Third, bladder emptying requires synchronous activation of all the smooth muscle of the bladder body, because if only part of the wall contracted, the uncontracted compliant areas would stretch and prevent the increase in pressure necessary for urine to be expelled through the urethra.

Anatomy of Hypogastric, Pelvic Splanchnic and Pudendal Nerve

The hypogastric nerve is a term for the transition between the superior hypogastric plexus and the inferior hypogastric plexus. The hypogastric nerve begins where the superior hypogastric plexus splits into a right and left plexus. Each of these divisions is

considered a hypogastric nerve. The hypogastric nerve continues inferiorly on its corresponding side of the vertebral body, where it descends into the pelvis to form inferior hypogastric plexus.

The superior hypogastric plexus (hypogastric plexus or presacral nerve) which receives contribution from T10 to L2 splanchnic nerves is a plexus of nerves situated on the vertebral bodies anterior to the bifurcation of the abdominal aorta [4].

The inferior hypogastric plexus (pelvic plexus) is a plexus of nerves that supplies the viscera of the pelvic cavity.The inferior hypogastric plexus is a paired structure, with each situated on the side of the rectum in the male, and at the sides of the rectum and vagina in the female [4].Contributions to the plexus include:

1. A continuation of the superior hypogastric plexus on either side, in the form of the hypogastric nerve.
2. Lumbar splanchnic nerves L3 to L4
3. Pelvic splanchnic nerves (from the second, third, and fourth sacral nerves) contribute parasympathetic efferent fibers to the plexus.

From these plexuses numerous branches are distributed to the viscera of the pelvis. The parasympathetic fibers which arise from pelvic splanchnic nerve (S2-S4) ascend from inferior hypogastric plexus, it is more usual for these parasympathetic fibers to ascend to the left-handed side of the superior hypogastric plexus and cross the branches of the sigmoid and left colic vessel branches, as these parasympathetic branches are distributed along the branches of the inferior mesenteric artery [4].

From these plexus numerous branches accompany the branches of the internal iliac artery which is the source for the middle rectal plexus, vesical plexus, prostatic plexus, and uterovaginal plexus [4].

Pelvic splanchnic nerves or Nervierigentes are splanchnic nerves that arise from sacral spinal nerves S2, S3, S4 to provide parasympathetic innervation to the pelvic viscera. The pelvic splanchnic nerves arise from the anterior rami of the sacral spinal nerves S2-S4 and enter the sacral plexus. They travel to their side's corresponding inferior hypogastric plexus, located bilaterally on the walls of the rectum [4].

From there, they contribute to the innervation of the pelvic and genital organs. The nerves regulate the emptying of the urinary bladder, control opening and closing of the internal urethral sphincter, influence motility in the rectum as well as sexual functions like erection. They contain both preganglionic parasympathetic fibers as well as visceral afferent fibers [4].

Pudendal nerve (S 2, 3, & 4) is the main nerve of the perineum. It carries sensation from the external genitalia of both sexes and the skin around the anus and perineum, as well the motor supply to various pelvic muscles, including the external urethral sphincter and the external anal sphincter [4].

Neural Control of the Lower Urinary Tract

Peripheral Nervous System

The lower urinary tract is innervated by three sets of peripheral nerves involving the parasympathetic, sympathetic, and somatic nervous systems. Pelvic parasympathetic

nerves arise at the sacral level of the spinal cord, excite the bladder detrusor, and relax the internal urethral sphincter. Lumbar sympathetic nerves inhibit the bladder detrusor and excite the internal urethral sphincter. Pudendal nerves excite the external urethral sphincter. These nerves contain afferent (sensory) as well as efferent axons [4].

Parasympathetic Pathways

Parasympathetic preganglionic neurons innervating the lower urinary tract are located in the lateral part of the sacral intermediate gray matter in a region termed the sacral parasympathetic nucleus [4]. Parasympathetic preganglionic neurons send axons through the ventral roots to peripheral ganglia, where they release the excitatory transmitter acetylcholine [4]. Parasympathetic postganglionic neurons in humans are located in the detrusor wall layer as well as in the pelvic plexus. This is an important fact to remember because patients with caudaequina or pelvic plexus injury are neurologically decentralized but may not be completely denervated. Caudaequina injury allows possible afferent and efferent neuron interconnection at the level of the intramural ganglia [4].

Muscarinic Mechanisms

There are at least five muscarinic receptor subtypes. Pharmacologically, M1, M2, and M3 receptor subtypes have been found in the human bladder. Stimulation of M3 receptors by acetylcholine induces calcium influx through L-type Ca2+ channels, as well as IP3 hydrolysis due to phospholipase C activation, resulting in the release of intracellular calcium, both of which contribute to a smooth muscle contraction. Muscarinic receptor subtype–mediated detrusor contractions shift from M3 to M2

receptor subtype has been reported in bladder muscle specimens from neurogenic bladder dysfunction patients.

Sympathetic Pathways

Sympathetic outflow from the lumbar spinal cord provides a noradrenergic excitatory and inhibitory input to the bladder and urethra [9]. Activation of sympathetic nerves induces relaxation of the bladder detrusor and contraction of the internal urethral sphincter, which contributes to urine storage in the bladder. The peripheral sympathetic pathways follow a complex route that passes through the sympathetic chain ganglia to the inferior mesenteric ganglia and then through the hypogastric nerves to the pelvic ganglia [4].

Adrenergic Mechanisms

β-Adrenergic stimulated relaxation is mediated through the activation of adenylatecyclase and the accumulation of cyclic AMP (cAMP). The β3-adrenergic receptor is the most highly expressed subtype among α- and β-adrenoceptor subtypes, and β3- receptor agonists are in clinical trial for treatment of detrusor overactivity. In the human, there is a predominant expression of α1D receptors present in the normal bladder, and the level of expression of α-adrenoceptor mRNA, which is considerably low compared with β3 adrenoceptors in normal bladders, was not increased in the bladder with outflow obstruction. Urethral tone and intra-urethral pressure are influenced by α-adrenergicreceptors. The α1A adrenoceptor is the major subtype in the prostate and

urethra, and all three α1 adrenoceptor subtypes (α1A, α1B, α1D) are present in blood vessels.

Somatic Pathways

The external urethral sphincter motor neurons are located along the lateral border of the ventral horn, commonly referred to as the Onuf nucleus [10]. Sphincter motor neurons also exhibit transversely oriented dendritic bundles that project laterally into the lateral funiculus, dorsally into the intermediate gray matter, and dorsomedially toward the central canal [10].

Afferent Pathways

Afferent axons in the pelvic, hypogastric, and pudendal nerves transmit information from the lower urinary tract to the lumbosacral spinal cord [10]. The primary afferent neurons of the pelvic and pudendal nerves are contained in sacral dorsal root ganglia (DRG), whereas afferent innervation in the hypogastric nerves arises in the lumbar DRG. The central axons of the DRG neurons carry the sensory information from the lower urinary tract to second-order neurons in the spinal cord [10].

Visceral afferent fibers of the pelvic and pudendal nerves enter the cord and travel caudally. Pelvic nerve afferents, which monitor the volume of the bladder and the amplitude of the bladder contraction, consist of myelinated (Aδ) and unmyelinated (C) axons. During neuropathic conditions and possibly inflammatory conditions, there is recruitment of C fibers that form a new functional afferent pathway that can cause urgency incontinence and possibly bladder pain [10].

Reflex Circuitry Controlling Micturition

Afferent pathway	Efferent pathways	Central pathway
Urine Storage Low-level vesical afferent activity (pelvic nerve)	External sphincter contraction (somatic nerves) Internal sphincter contraction (sympathetic nerves) Detrusor inhibition (sympathetic nerves) Ganglionic inhibition (sympathetic nerves) Sacral parasympathetic outflow inactive	Spinal reflexes
Micturition High-level vesical afferent activity (pelvic nerve)	Inhibition of external sphincter activity Inhibition of sympathetic outflow Activation of parasympathetic outflow to the bladder Activation of parasympathetic outflow to the urethra	Spinobulbospinal reflex

Multiple reflex pathways organized in the brain and spinal cord mediate coordination between the urinary bladder and the urethra. The central pathways controlling lower urinary tract function are organized as simple on-off switching circuits that maintain a reciprocal relationship between the urinary bladder and the urethral outlet [4].

Spinal and Supraspinal Pathways Involved in the Micturition Reflex

Spinal Cord

In the spinal cord, afferent pathways terminate on second-order interneuron's that relay information to the brain or to other regions of the spinal cord, including the preganglionic and motor nuclei. Because disynaptic or polysynaptic pathways, but not monosynaptic pathways, mediate bladder, urethral, and sphincter reflex, interneuronal mechanisms must play an essential role in the regulation of lower urinary tract function [4].

The micturition reflex can be modulated at the level of the spinal cord by interneuronal mechanisms activated by afferent input from cutaneous and striated muscle targets. Micturition reflex can also be modulated by inputs from visceral organs [4]. Stimulation of afferent fibers from various regions (anus, colon-rectum, vagina, uterine cervix, penis, perineum, pudendal nerve) can inhibit the firing of sacral interneurons evoked by bladder distention [4]. This inhibition may be a result of presynaptic inhibition at primary afferent terminals or be due to direct postsynaptic inhibition of the second-order neurons.

Direct postsynaptic inhibition of bladder preganglionic neurons can also be elicited by stimulation of somatic afferent axons in the pudendal nerve or visceral afferents from the distal bowel [4]. Suppression of detrusor overactivity in patients by sacral root stimulation may reflect, in part, activation of the afferent limb of these visceral bladder and somatic-bladder inhibitory reflexes [12].

Pontine Micturition Center and Brainstem Modulatory Mechanisms

Various studies indicate that the micturition reflex is normally mediated by a spinobulbospinal reflex pathway passing through relay centers in the brain [4]. The dorsal pontinetegmentum has been firmly established as an essential control center for micturition in normal subjects. First described by Barrington (1921) [13], it has subsequently been called the Barrington nucleus, the pontine micturition center or the M region because of its medial location [14].

Neurons in the PMC provide direct synaptic inputs to sacral preganglionic neurons as well as to GABAergic neurons in the sacral dorsal commissure region [14]. The former neurons carry the excitatory outflow to the bladder, whereas the latter neurons are thought to be important in mediating an inhibitory influence on external urethral sphincter motor neurons [14]. In addition to providing axonal inputs to the locus ceruleus and the sacral spinal cord, neurons in the pontine micturition center (PMC) also send axon collaterals to the paraventricular thalamic nucleus, which is thought to be involved in the limbic system modulation of visceral behavior [15]. Some neurons in the PMC also project to the periaqueductal gray region (PAG), which regulates many visceral activities as well as pain pathways [16]. Thus neurons in the PMC communicate with multiple supraspinal neuronal populations that may coordinate micturition with other functions of the organism. Although the circuitry in humans is uncertain, brain imaging studies have revealed increases in blood flow in this region of the pons during micturition [14]. This

change presumably reflects increases in neuronal activity. Thus the PMC appears critical for micturition across species [14].

Projections from neurons in the lateral pons (L-region or nucleus locus subcoeruleus) terminate rather selectively in the sphincter motor nucleus to enhance the contraction of pelvic floor muscle and increased urethral pressure during the storage phase in cats, providing evidence that this area in the lateral pons functions as "the pontine continence center" [17]. Because of these reciprocal connections, the PMC can promote bladder–sphincter synergy [17].

In the midbrain, there is increasing evidence that the mesencephalic PAG, which directly receives information of bladder fullness by ascending spinal tract neurons and sends projections to the PMC, seems to play an important role in the integration of the micturition reflex [14]. The PAG appears to function as a relay center between the PMC and spinal cord neurons that are critically involved in the excitatory and inhibitory control of the micturition reflex [14].

Central Pathways That Modulate the Micturition Reflex

Voluntary control of voiding is dependent on connections between the frontal cortex and the septal-preoptic region of the hypothalamus, as well as on connections between the paracentral lobule and the brainstem. Lesions to these areas of cortex appear to directly increase bladder activity by removing cortical inhibitory control [4].

Physiology of micturition

The micturition reflex is a finely tuned, coordinated neuromuscular event characterized by an orderly physiologic sequence. The two functions of the lower urinary tract are the storage of urine within the urinary bladder and the timely expulsion of urine from the urethra. The storage and expulsion of urine are part of a complex neurophysiologic function that involves autonomic and somatic nervous systems. Function is controlled by reflex pathways, which are further modulated by central voluntary control.

The micturition cycle involves two relatively discrete processes:

(1) Bladder filling and urine storage, and (2) Bladder emptying or voiding.

Bladder filling and urine storage require:

1. Accommodation of increasing volumes of urine at low intravesical pressure (normal compliance) and with appropriate sensation.
2. A bladder outlet that is closed at rest and remains so during increases in intra-abdominal pressure.
3. Absence of involuntary bladder contractions (detrusor over activity)

Bladder emptying/voiding requires:

1. A coordinated contraction of the bladder smooth musculature of adequate magnitude and duration.

2. A concomitant lowering of resistance at the level of the smooth and striated sphincter (no functional obstruction)
3. Absence of anatomic (as opposed to functional) obstruction

Bladder response during filling:

The normal adult bladder response to filling at a physiologic rate is an almost imperceptible change in intravesical and detrusor pressure. During at least the initial stages of bladder filling, after unfolding of the bladder wall from its collapsed state, this high compliance (volume/pressure) of the bladder is due primarily to its elastic and viscoelastic properties. Elasticity allows the constituents of the bladder wall to stretch to a certain degree without any increase in tension. Viscoelasticity allows stretch to induce a rise in tension followed by a decay ("stress relaxation") when the filling (stretch stimulus) slows or stops. The viscoelastic properties are considered to be primarily due to the characteristics of the extracellular matrix in the bladder wall [18].The viscoelastic properties of the stroma (bladder wall less smooth muscle and epithelium) and the urodynamically relaxed detrusor muscle thus account for the passive mechanical properties and normal bladder compliance seen during filling. The main components of the stroma are collagen and elastin. In the usual clinical setting, filling cystometry seems to show a slight increase in intravesical pressure, but this pressure rise is a function of the fact that cystometric filling is carried out at a greater thanphysiologicrate and that, at physiologic filling rates, there is essentially no rise in bladder pressure until bladder capacity is reached [19].

When the collagen component of the bladder wall increases, compliance decreases. This can occur with chronic inflammation, bladder outlet obstruction, neurologic decentralization, and various other types of injury. Bladder muscle hypertrophy, which can result from outlet obstruction, can also result in decreased compliance because it is said to be less elastic than normal detrusor; it also can synthesize increased amounts of collagen. Once decreased compliance has occurred because of a replacement by collagen of other components of the stroma, it is generally unresponsive to pharmacologic manipulation, hydraulic distension, or nerve section. Most often, under those circumstances, augmentation cystoplasty is required to achieve satisfactory reservoir function [4].

At a certain level of bladder filling, spinal sympathetic reflexes facilitatory to bladder filling / storage are clearly evoked in animals, a concept developed over the years by DeGroatet al who has also cited indirect evidence to support such a role in humans [4]. This inhibitory effect is thought to be mediated primarily by sympathetic modulation of cholinergic ganglionic transmission.

Through this reflex mechanism, two other possibilities exist for promoting filling/ storage. One is neutrally mediated stimulation of the predominantly α -adrenergic receptors (α1) in the area of the smooth sphincter, the net result of which would be to cause an increase in resistance in that area. The second is neutrally mediated stimulation of the predominantlyβ-adrenergic receptors (β3 inhibitory) in the bladder body smooth musculature, which would cause a decrease in bladder wall tension. Direct inhibition of

detrusor motor neurons in the sacral spinal cord during bladder filling related to increased afferent pudendal nerve activity generated by receptors in the striated sphincter. Good evidence also seems to exist to support an inhibitory effect of other neurotransmitters (e.g. glycine, gamma ammonia butyric acid [GABA], opioids, purines, and the noradrenergic system) on the micturition reflex at various levels of the neural axis [4].

Outlet response during filling

There is a gradual increase in urethral pressure during bladder filling, contributed to at least by the striated sphincteric element and perhaps by the smooth sphincteric element as well. The rise in urethral pressure seen during the filling/storage phase of micturition can be correlated with an increase in efferent pudendal nerve impulse frequency and in electromyographic activity of the striated sphincter. This constitutes the efferent limb of a spinal somatic reflex, the so called guarding reflex, which results in a gradual increase in striated sphincter activity during normal bladder filling and storage [20].The passive properties of the urethral wall certainly deserve mention because these undoubtedly play a role in the maintenance of continence. Urethral wall tension develops within the outer layers of the urethra; however, urethral pressure is a product not only of the passive characteristics of the elastic, collagenous, and vascular components of the urethral wall because this tension must be exerted on a soft or plastic inner layer capable of being compressed to a closed configuration-the “filler material” representing the submucosal portion of the urethra [21].

Voiding with a normal bladder contraction:

Although many factors are involved in the initiation of micturition, in adults it is intravesical pressure producing the sensation of distension that is primarily responsible for the initiation of normal voluntarily induced emptying of the lower urinary tract. Although the origin of the parasympathetic neural outflow to the bladder, the pelvic nerve, is in the sacral spinal cord, the actual coordinating centre for the micturition reflex in an intact neural axis is in the brainstem. The complete neural circuit for normal micturition includes the ascending and descending spinal cord pathways to and from this area and the facilitatory and inhibitory influences from other parts of the brain, particularly the cerebral cortex. The final step in voluntarily induced micturition involves inhibition of the striated sphincter and an inhibition of all aspects of any spinal sympathetic reflexes evoked during filling. Efferent parasympathetic pelvic nerve activity is ultimately what is responsible for a highly coordinated contraction of the bulk of the bladder smooth musculature [22].

Outlet response during emptying

A decrease in outlet resistance occurs with adaptive shaping or funnelling of the relaxed bladder outlet. Besides the inhibition of any continence promoting reflexes that have occurred during bladder filling, the change in outlet resistance may also involve an active relaxation of the smooth sphincter area through a non-adrenergic non-cholinergic (NANC) mechanism, proposed to be mediated by nitric oxide. The adaptive changes that

occur in the outlet are probably also due at least in part to the anatomic interrelationships of the smooth muscle of the bladder base and proximal urethra. Longitudinal smooth muscle continuity would promote shortening and widening of the proximal urethra during a coordinated emptying bladder contraction. Other reflexes that are elicited emptying bladder contraction and by the passage of urine through the urethra may reinforce and facilitate complete bladder emptying. Superimposed on these autonomic and somatic reflexes is complex, modifying supraspinal inputs from other central neuronal networks. These facilitatory and inhibitory impulses, which originate from several areas of the nervous system, allow the full conscious control of micturition in the adult [22].

Urinary Continence during Abdominal Pressure Increases:

During voluntarily initiated micturition, the bladder pressure becomes higher than the outlet pressure and certain adaptive changes occur in the shape of the bladder outlet with consequent passage of urine into and through the proximal urethra. One could reasonably ask, why do such changes not occur with increases in intravesical pressure that are similar in magnitude but that are produced only by changes in intro- abdominal pressure such as straining or coughing? [23].

First, a coordinated bladder contraction does not occur in response to such stimuli, clearly emphasizing the fact that increases in total intravesical pressure are by no means equivalent to emptying ability. Secondly, for urine to flow into and through the proximal urethra in an individual who does not have sphincterincontinence, there must be (1) an

increase in intravesical pressure that is primarily a product of a coordinated, neutrally mediated bladder contraction and that is (2) associated with characteristics tension and conformational changes in the bladder neck and proximal urethra [23].

Assuming that the bladder outlet is competent at rest, a major factor required for the prevention of urinary leakage during increases in intra-abdominal pressure is that there is at least equal pressure transmission to the proximal urethra (the midurethra , as well as in the female) during such activity. The phenomenon was first described by Enhorning (1961) and has been confirmed in virtually every urodynamic laboratory since that time. Failure of this mechanism is an invariable correlate of effort- related urinary incontinence in the female and male. The urethral closure pressure, indicating that active muscular function related to a reflex increase in striated sphincter activity or other factors that increase urethral resistance is also involved in preventing such leakage [23].

Sensory Aspects

Most of the afferent input from the bladder and urethra reaches the spinal cord throughthe pelvic nerve and dorsal root ganglia and some through the hypogastric nerve. Afferent input from the striated muscle of the sphincter and pelvic floor travels in the pudendal nerve. The most important afferents for initiating and maintaining normal micturition are those in the pelvic nerve, relaying to the sacral spinal cord. These convey impulses from tension, volume, and nociceptive receptors located in the serosal, muscle, and urothelial and suburothelial layers of the bladder and urethra [24].

Pathophysiology of abnormalities of filling/ storage and emptying/voiding:

Filling/Storage Failure

Absolute or relative failure of the bladder to fill with and store urine adequately results from the bladder overactivity (involuntary contraction or decreased compliance), decreased outlet resistance, heightened or altered sensation, or a combination. The treatment of filling/storage abnormalities is directed toward inhibiting bladder contractility, decreasing sensory output, or mechanically increasing bladder capacity; and /or toward increasing outlet resistance, the latter either continuously or just during increases in intra-abdominal pressure [25].

Emptying/Voiding Failure

Absolute or relative failure to empty the bladder results from decreased bladder contractility (a decrease in magnitude or duration), increased outlet resistance, or a combination. The treatment of emptying failure generally consists of maneuvers to increase intravesical / detrusor pressure, facilitate the micturition reflex, decrease outlet resistance, or a combination. If other means fail or are impractical, intermittent (or continuous) catheterization is an effective way to circumvent emptying failure [25].

Lower urinary tract symptoms [26]

Lower urinary tract symptoms are divided into three groups, storage, voiding, and post-micturition symptoms.

Storage symptoms are experienced during the storage phase of the bladder and include daytime frequency and nocturia.

1. **Increased daytime frequency** is the complaint by the patient who considers that he/she voids too often by day

2. **Nocturia** is the complaint that the individual has to wake at night one or times to void;

3. **Urgency** is the complaint of a sudden compelling desire to pass urine which is difficult to defer

4. **Urinary incontinence** is the complaint of any involuntary leakage of urine. Urinary leakage may need to be distinguished from sweating or vaginal discharge.

    a. **Stress urinary incontinence** is the complaint of involuntary leakage on effort or exertion, or on sneezing or coughing.

    b. **Urge urinary incontinence** is the complaint of involuntary leakage accompanied by or immediately preceded by urgency.

    c. **Mixed urinary incontinence** is the complaint of involuntary leakage associated with urgency and also with exertion, effort, sneezing, or coughing.

    Enuresis means any involuntary loss of urine. If it is used to denote incontinence during sleep, it should always be qualified with the adjective “nocturnal.”

Nocturnal enuresis is the complaint of loss of urine occurring during sleep.

Continuous urinary incontinence is the complaint of continuous leakage.

Other types of urinary incontinence may be situational for example the report of incontinence during sexual intercourse, or giggle incontinence.

5. **Bladder sensation** can be defined during history taking, by five categories.

    a. **Normal**: the individual is aware of bladder filling and increasing sensation up to a strong desire to void.
    b. **Increased**: the individual feels an early and persistent desire to void.
    c. **Reduced**: the individual is aware of bladder filling but does not feel a desire to void.
    d. **Absent**: the individual reports no sensation of bladder filling or desire to void.
    e. **Non-specific**: the individual reports no specific bladder sensation but may perceive bladder filling as abdominal fullness, vegetative symptoms, or spasticity.

Voiding symptoms are experienced during the voiding phase.

1. **Slow stream** is reported by the individual as his or her perception of reduced urine flow, usually compared to previous performance or in comparison to others.
2. **Splitting or spraying** of the urine stream may be reported.

3. **Intermittent stream (intermittency)** is the term used when the individual describes urine flow which stops and starts, on one or more occasions, during micturition.
4. **Hesitancy** is the term used when an individual describes difficulty in initiating micturition resulting in a delay in the onset of voiding after the individual is ready to pass urine.
5. **Straining** to void describes the muscular effort used to initiate, maintain or improve the urinary stream.
6. **Terminal dribble** is the term used when an individual describes a prolonged final part of micturition, when the flow has slowed to a trickle/dribble.

Post micturition symptoms are experienced immediately after micturition.

1. **Feeling of incomplete emptying** is a self-explanatory term for a feeling experienced by individual after passing urine.
2. **Post micturition dribble** is the term used when an individual describes the involuntary loss of urine immediately after he/she has finished passing urine, usually after leaving the toilet in men, or after rising from the toilet in women [26].

Urodynamics

Urodynamics refers to a group of tests used to assess function of the lower urinary tract by measuring various aspects of urine storage and emptying. Some specific types of urodynamic testing are:

1. Cystometry (or cystometrogram) evaluates bladder function by measuring pressure and volume of fluid in the bladder during filling, storage, and voiding.
2. Uroflowmetry measures the rate of urine flow.
3. Urethral pressure profile tests urethral function.
4. Leak point pressure determines the bladder or abdominal pressure when leakage occurs due to increased abdominal pressure (valsalva or cough) to assess urethral resistance.
5. Post void residual urine.

The purpose of urodynamic testing is to aid in understanding physiology mechanisms of lower urinary tract dysfunction, thereby improving the accuracy of diagnosis and facilitating targeted treatment.

Efficacy

Clinical evaluation with urodynamics may lead to a more accurate diagnosis of incontinence type [27]. A meta-analysis including several thousand women reported the symptom of stress incontinence, when compared against an urodynamic diagnosis, was 91% sensitive, but only 51 % specific in diagnosis pure stress incontinence [27]. For urgency incontinence, history alone was 73 %sensitive, but only 55 % specific. In a chart review study of women with urinary incontinence, the probability of a change in treatment plan with, versus without, urodynamic data was 27% [28].

In contrast, a Cochrane review concluded there were insufficient data from randomized studies to determine whether treatment of urinary incontinence according to

a urodynamic-based diagnosis was more effective than treatment based upon history and examination alone [29].

Cystometry

Filling cystometry is the method by which the pressure/ volume relationship of the bladder is measured during bladder filling. It is used to assess detrusor activity and bladder sensation, capacity, and compliance. Cystometry can be done with one channel measuring bladder pressure alone or with an additional channel that simultaneously measures abdominal pressure through the rectum or vagina.

The advantage of the multichannel test is that it can discriminate between changes in abdominal versus bladder pressure by electronically subtracting the abdominal component from intravesical pressure.

Only a multichannel test can show that a rise in bladder pressure is due to detrusor contraction rather than testing of the abdominal wall. This information is important for avoiding a false impression of detrusor over activity. As an example, detrusor over activity during single channel cystometry is not reproducible by multichannel test result is the most reliable for evaluating women with incontinence [30].

Some urodynamic machines can also record urethral pressure with a second sensor on the bladder catheter. This allows electronic calculation of urethral closure pressure, which is the difference between urethral and bladder pressures. Leakage usually occurs when bladder pressure exceeds urethral pressure. However, the clinical value of this measurement has not been proven.

Indications

The most common reason to do cystometry is to distinguish detrusor overactivity (involuntary contractions of the bladder) from incontinence (leakage due to an increase in abdominal pressure). The test can also identify patients with abnormalities of bladder sensation and mixed incontinence.

Modification of cystometry

1. VideoUrodynamics– This technique is similar to conventional cystometry, but with the addition of a radio-opaque filling medium, video recorder, and X-ray equipment. Video urodynamics may offer information beyond conventional urodynamics in complicated neurologic conditions, but most authorities believe it is seldom indicated [31]. The disadvantages are radiation exposure, cost, and the extra technical support required.
2. Ambulatory Urodynamics – Ambulatory testing can be performed by using commercially available systems in which an ambulatory patient wears an indwelling catheter connected to a microcomputer. The advantage over conventional cystometry is that the home environment reflects the patient's functional status more realistically than the laboratory. Several studies have shown the ambulatory urodynamics are more sensitive than conventional urodynamics in diagnosing detrusor overactivity [32].

Uroflowmetry

Uroflowmetry measures urine volume voided over time. It can be done with or

without a pressure- flow study, which measures detrusor pressure during voiding.

Indications - Abnormal voiding is much more common in men than in women. Nevertheless, uroflowmetry is usually done as part of a complete urodynamic evaluation in women. It can be useful in clinical situations, such as

1. Frequency, urgency, and urgency incontinence as some of these patients have outlet obstruction.
2. Voiding difficulty, hesitancy, or difficulty maintaining the urine stream, which can also be due to outlet obstruction (from previous pelvic surgery or urethral kinking with anterior vaginal wall prolapsed) or weak detrusor (as in neurologic disease).
3. Planned pelvic surgery because poor uroflow may be a predictor of postoperative voiding difficulty after radical pelvic surgery. This information allows for preoperative counselling or training in self-catheterization; however, the predictive value of uroflowmetry is controversial of postoperative voiding difficulty [33].

Technique

After the physician and nurse have left the room, the patient voids into a special commode that measures the amount and rate of urine flow. Although there are no universally accepted standards, a normal uroflow test consists of:

1. Volume greater than 200 ml over 15 to 30 seconds
2. Maximum flow rate greater than 15 ml/sec (after correction for artefact)
3. Continuous single curve (as opposed to short spikes) of flow

   Maximum flow rates < 15ml/sec may indicate obstruction, detrusor weakness

or significant valsalva effort during voiding [34]. An acontractile detrusor is unable to initiate a contraction and will lead to overflow incontinence.

Some patients are unable to void normally in the artificial conditions of the urodynamics laboratory; thus, it is important to ask the patient if she feels that her void was typical for her, especially if an abnormal pattern is observed. If the patient feels the test was atypical, it should be repeated.

Pressure Flow Study

A pressure-flow study measures both bladder pressure and urinary flow. The purpose of a pressure-flow rate is to determine the mechanism of abnormal voiding revealed by a low rate on uroflowmetry. If the detrusor pressure is high (>50 cm H2O), urethral overactivity (detrusor-sphincter dysynergia) or urethral obstruction (structure or tumor) may be present. The most common cause of obstructed voiding in females is prolapse of the anterior vaginal wall, which can cause kinking of the pressure-flow study in women, is to determine the cause of voiding dysfunction after surgery [35].

One study that compared women with bladder outlet obstruction to normal controls reported a maximum detrusor pressure >25cm H2O gave the greatest sensitivity and specificity, >60% for detection of obstruction [36]. An uroflow, 12ml/sec gave equal sensitivity, specificity and accuracy (68 %) for predicting obstruction.

A pressure flow study can be done as an extension of cystometry when catheters are already in place to measure detrusor and abdominal pressure. The patient voids around the urethral catheter into a commode, which simultaneously measures uroflow.

If the patient has an acontractile or poorly contractile detrusor, she will have a low flow rate (<15 ml/ sec maximum flow) and little or no rise in true detrusor pressure. Voiding occurs mainly by abdominal straining. Since many patients use valsalva to aid voiding, a rise in abdominal pressure alone does not indicate a problem. Detrusor acontractility is often due to a neurologic abnormality, such as spinal cord injury or multiple sclerosis, but it may be idiopathic.

Test results are difficult to assess because of the difficulty of voiding around the catheter, as well as the artificial surroundings of the urodynamics laboratory. There are no accepted standards for the method and interpretation of pressure flow studies.

Urethral Pressure Profile

The pressure in the urethra should be equal to or greater than the bladder pressure during bladder filling. When the bladder and urethra are in their proper anatomic location, increases in intra-abdominal pressure will also increase urethral pressure, thereby preventing leakage. Low urethral pressure can be associated with incontinence and is related to aging, hypo-estrogenic state, multi-parity, and previous significant urogyneological operations.

The urethral pressure profile (UPP) measures the intraluminal pressure along the entire length of the urethra with the bladder at rest. A special catheter with an intravesical and an intraureethral pressure transducer and side openings near its tip is perfused and slowly withdrawn from the bladder. The transducers are usually brought out to the external urethral meatus at a rate of 1 mm/sec by a mechanical pulling device. The

pressure necessary to maintain a constant flow provides an assessment of the activity of the urethral wall. The clinical purpose of the urethral pressure profile (UPP) is to help distinguish intrinsic sphincter deficiency from genuine stress incontinence [37].

Leak Point Pressure

Abdominal leak point pressure refers to the intravesical pressure at which urine leakage occurs due to increased abdominal pressure in the absence of a detrusor contraction. The pressure can be produced by valsalva or cough. Unlike the urethral pressure profile, the leak point pressure reflects urethral function in the dynamic situation that produces incontinence.

Leak point pressure is used to assess intrinsic sphincter function. It is more reliable than the urethral pressure profile for diagnosis of intrinsic sphincter deficiency [38].

Post-void Residual Volume

This measurement is made either by straight catheterization or by bladder ultrasound. Small portable ultrasounds specifically for post-void residual measurement are available. A normal patient should have the ability to void at least 80% of the total bladder volume and have residual urine <50 mL immediately after voiding. High residual urine on repeated determinations indicates outlet obstruction or poor detrusor contractility.

Electro-physiologic testing

Various electro-physiologic tests has also been used to evaluate lower urinary tract

dysfunction. They have been employed mainly in the context of research, rather than clinical care.

1. Electromyography (EMG) is the study of electrical potentials generated by depolarization of muscle. This can be done with surface electrodes, a needle inserted into urethral sphincter, a monopolar electrode inside the urethra, a concentric needle inserted into the urethral sphincter, or a single fiber to record action potentials from individual muscle fibers.
2. Nerve conduction studies (NCS) examine nerve conduction rates from astimulus to a receptor at a distal site. Pudendal nerve terminal motor latency and perinealnerve terminal motor latency (the time elapsed between initiating an impulse and its arrival at nerves end) can be determined.
3. Evoked potentials are the sum of potentials recorded in central nervous tissue (spinal cord or cortex) after stimulation of a peripheral site such as the perineum.

Pitfalls in urodynamic testing:

Numerous pitfalls in urodynamic testing limit its value [39]. Some fundamental problems include:

1. Lack of standardization of technical details, such as patient position, type of pressure sensor, and filling rate. These variables significantly affect results.
2. The artificial situation of the urodynamic laboratory, which produces non physiologic results in some patients.
3. Use of a transurethral catheter can unmask stress incontinence [40].

4. Inconsistent reproducibility of test results in the same patient [41].
5. The wide range of physiologic values in normal, asymptomatic patients [42].
6. The absence of a specific abnormality during urodynamic testing does not exclude its existence, and not all abnormalities found during urodynamics are clinically significant.

Radical Hysterectomy

History of Radical Hysterectomy

Radical hysterectomy was initially developed as a treatment for cervical cancer due to the absence of other modalities of treatment. John Clark performed the first radical hysterectomy at John Hopkins Hospital in 1891[43]. Clark and Ries noted the spread of cancer to the tissues and lymph nodes beyond the limits of excision of the standard hysterectomy. Each of them developed a more radical hysterectomy, removing more of the broad ligament, vagina and associated pelvic lymph nodes [43].

In 1898, Ernst Wertheim, a Viennese physician, performed the first full extended radical operation for cervical cancer. He developed radical total abdominal hysterectomy with removal of the uterus, tissues surrounding the upper vagina, pelvic lymph nodes and the parametrium [43]. Wertheim did not routinely perform lymphadenectomy, removing only clinical enlarged or suspicious nodes. When he reported the outcomes of treatment of the first 270 patients, the mortality rate figure was at 18 % and the morbidity rate was 31% [44].

The more radical extension of vaginal hysterectomy was developed by Karl Schuchardt of Gottingen and particularly FridrichSchauta of Vienna [43]. In 1901, Schauta described radical vaginal hysterectomy and reported a lower operative mortality rate than the one of the abdominal approach [44]. The inability to perform adequate pelvic lymphadenectomy in the vaginal procedure resulted in the sway of the abdominal approach. As a result of the overall, still high mortality rates, radiation therapy replaced surgery as the treatment of the choice for cervical cancers [44].

In 1919, Latzko developed paravesical and pararectal space and divided the cardinal ligament wider than Wertheim hysterectomy [45]. In 1921, HidekazuOkabayashi proposed his technique of radical abdominal hysterectomy (the same technique of Latzko operation, but different management of vesicuouterine ligament), but more as a modification of the surgery performed by his teacher Prof. ShouheiTakayama. The principles of Okabayashi procedure (i) usage of avascular planes, (ii) cleavage of recto-uterine ligaments before managing the vascular web and (iii) dissecting the ureter at the end of the procedure because of the increased mobility of the uterus[45].

In 1941, Mibayashi did total resection of the cardinal ligament with blood vessels of the internal iliac artery and vein (ligation of obturator, hypogastric, internal pudendal and inferior gluteal artery and vein) [46].

In 1954, Joe Vincent Meigs re-popularized the surgical approach when he developed a modified Wertheim operation with removal of the pelvic nodes [47]. Meigs reported a survival rate of 75% for stage I disease and demonstrated an operative

mortality rate of 1%. In 1960, Kobayashi described radical hysterectomy with preservation of the pelvic splanchnic nerves [48]. There have been several modifications of the operative technique, and in recent years laparoscopy has increasingly been employed in the management of early-stage cervical cancer.

Total laparoscopic radical hysterectomy was done in 1990 by Canis M [49] and in 1992 by Nezhat C.R [50].The first robotic-assisted radical hysterectomy was reported by Sert and Abeler in 2006[51]. Robotic surgery, a new tool in surgical armamentarium is designed to remove the limitations of traditional laparoscopy. The advantages of robotic radical hysterectomy are improved visualization, improved dexterity and limited fatigue and physical discomfort.

Indications for Radical Hysterectomy

Radical hysterectomy is performed as a primary therapy for

1. Stage I B or II A cervical cancer
2. Selected patients with stage II adenocarcinoma of endometrium with suspected endocervical involvement in whom radical surgery seems feasible
3. Upper vaginal carcinoma, uterine or cervical sarcomas, and other rare malignancies confined to the area of cervix, uterus, and/or upper vagina

Salvage therapy for women with cervical cancer who have been treated with irradiation and subsequently develop a small central pelvic recurrence or have a small central area of persistent disease. In these cases, the procedure may offer curative salvage treatment as an alternate to pelvic exenteration.

Identification of Basic Components of Surgical Classification:

There are two common measures of the outcome of the radical hysterectomy. First, some adverse effects such as bladder dysfunction correlate with anatomical extent of resection and nerve preservation when the pelvic autonomic nerves are threatened as a result of the extent of resection. Second, curative effect of surgery correlates with anatomical extent of resection, but needs documentation of the balance of benefits and risks associated with the procedure. Furthermore, combination of radiotherapy or chemotherapy, or both, with radical hysterectomy might negate any real difference in the curative effect of the extent of hysterectomy while adversely affecting the frequency of complications [52].

A classification is not a description of a technique. It is designed to establish the surgical template, not the way the surgeon achieves the goal. In 1974, Piver, Rutledge and Smith [53]and in 2008, European Organization for research and Treatment of Cancer – Gynecological Cancer Group (EORTC–GCG)and Querleu and Morrowclassified radical hysterectomy [54]. However, it should include general technical guidelines when they are crucial to the success or safety of the operation.

Classification of radical hysterectomy according to Piver, Rutledge and Smith [53]

Type I: Extra fascial hysterectomy

Deflection and retraction of the ureters without dissection of the ureteral bed

Uterine artery, uterosacral ligament and cardinal ligament are not removed

No vaginal cuff removed.

TYPE II: Modified radical hysterectomy

Ureters are freed from the paracervical position but are not dissected out of the pubovesicle ligament

Uterine arteries divided just medial to the ureter

Uterosacral ligaments resected midway between the uterus and their sacral attachments

Medial half of the cardinal ligaments removed

Upper one-third of the vagina removed

Elective pelvic lymphadenectomy

TYPE III: Classical Meigs' radical hysterectomy

Complete dissection of the ureter from the pubovesicle ligament to entry in the bladder except a small lateral part so that the superior vesicle artery is conserved

Uterine artery divided at origin from the internal iliac artery

Uterosacral ligaments resected at their sacral attachments

Cardinal ligaments resected at the pelvic wall

Upper half of the vagina removed

Routine pelvic lymphadenectomy

TYPE IV: More radical than type III in the three aspects:

Complete dissection of the ureter from the pubovesical ligament

The superior vesicle artery is sacrificed

Upper 3 quarters of the vagina removed

TYPE V: More radical than type IV:

Excision of involved portion of distal ureter or bladder and re-implantation of ureter into the bladder

Classification of radical hysterectomy according to Querleu and Morrow, [52]

Type A

Extrafascial hysterectomy

Visualisation and /or palpation of ureters without dissection of the ureteral bed

Uterine artery, uterosacral ligament and cardinal ligament are not transacted at a distance from the uterus

Minimal vaginal cuff removed (<10mm)

Type B

Ureters are unroofed and rolled laterally

Partial removal of uterosacral and vesicouterine ligaments

Transaction of the paracervix at the level of the ureteral tunnel

At least 10 mm of the vagina from the cervix or tumor is resected

Type B1: without removal of the lateral paracervical lymph nodes

Type B2: with additional removal of lateral paracervical lymph nodes

Type C

Ureters are completely mobilized

Transection of the uterosacral ligament at the rectum

Transection of the vesicouterine ligament at the bladder

Complete transection of the paracervix

15 – 20 mm of the vagina from the cervix or the tumor and the corresponding paracolpos is resected routinely

Type C1: with preservation of autonomic nerves

Type C2: without preservation of autonomic nerves

Type D

Type D1: resection of the entire paracervix at the pelvic side wall together with the hypogastric vessels, exposing the roots of the sciatic nerves

Type D2: type D1 plus resection of the entire paracervix at the pelvic side wall the hypogastric vessels and adjacent fascial or muscular structures

For all types, lymphadenectomy is described separately according to four levels.

L 1 - external and internal iliac nodes

L 2 – common iliac nodes

L 3 – aortic inframesentric nodes

L 4 – aortic infrarenal nodes

Nerve sparing technique of radical hysterectomy [55,56]

The nerve sparing technique, by laparotomic or laparoscopic route, includes 4 main steps:

1. The preservation of the superior hypogastric plexus at the level of the presacral area during the presacral lymphadenectomy;

2. The preservation of the hypogastric nerve dorsal to the ureter and lateral to the utero-sacral ligament during the section of the utero-sacral ligaments;

3. The preservation of the inferior hypogastric plexus during the section of the cardinal ligament, since the plexus lie dorsal to the parametrial vessels at the level of the deep uterine vein; and

4. The preservation of the bladder branch during the section of the deep layer of the cervico-vesical ligament.

Two main approaches have been proposed:

1. The identification of the hypogastric nerve followed by the section of the parametria medial to the nerve [55], this obtains a type 2;
2. The identification, clipping and section of the parametrial vessels at the level of the pudendal vessels, followed by the identification of the hypogastric nerve near to the utero-sacral ligaments and its separation from the fibrous part of the cardinal ligament; this technique obtains a type 3-4 [56].

Intraoperative and Postoperative Urological Complications of Radical Hysterectomy

With meticulous surgery, intra-operative injury to the urinary tract during radical hysterectomy is rare. The incidence of the following complications depends on the stage of the disease and the radicality of the procedure [57]

1. **Intra-operative injury of the bladder and ureters:** Almost in all cases of bladder or ureteral injury, the disease was relatively more invasive, for example

stage II B with cancer reaching the bladder or ureter, which are then sutured directly or require ureteral re-implantation [57]

2. **Postoperative fistula formation:** Radical hysterectomy requires the dissection of the entire pelvic course of the ureter, which may result in the interruption of several arterial branches that provide its blood supply [58]. Most fistulas result from postoperative ischemia, not from injury [57]. The incidence of vesicovaginal and ureterovaginal fistula formation about 0.6% - 6.6% has been reported. The fistulas usually appear between $12^{th} – 14^{th}$ postoperative days. The most common symptom exhibited by patients who developed ureterovaginal fistula is spontaneous leakage of urine from the vagina. The ureterovaginal fistula is usually located in the lower third of the ureter. One must wait for two to six months to repair the fistula. Based on experience, waiting for at least six months after operation has resulted in not only spontaneous healing but also more successful repair [58].
3. **Ureteric stricture formation** is also a potential hazard following radical hysterectomy, and should also be borne in mind when follow-up of patients treated with radical hysterectomy [59].

The Functional Alteration of Urinary Bladder and Urethra after Radical Hysterectomy

Postoperative lower urinary tract dysfunction is common among patients who undergo radical hysterectomy. Sensory loss and voiding disorders are the primary

complaints. These complications relate to nerve damage to the pelvic plexus and pelvic organs. During radical hysterectomy, parasympathetic denervation may occur if the surgeon strips the sacral nerves bilaterally or excises a major portion of the paracervical and paravaginal webs of retroperitoneal tissue. The sympathetic nerves are also vulnerable to injury during dissection of bilateral lymph nodes [60].

The pelvic plexus formed by the confluence of pelvic parasympathetic nerves with sympathetic hypogastric nerves, and also contains ganglia where parasympathetic and sympathetic nerves interact synchronously [61]. With nerve trunks emanating from S2-4, the pelvic plexus occupies the area of the inferior segment of the cardinal ligament. The urethra and bladder are connected to the terminal branches of the pelvic plexus [62].

Impaired micturition after a radical hysterectomy may result from altered modulation by the pelvic plexus and the resulting motor and sensory impairment of the detrusor [63]. Impaired detrusor contraction may be secondary to improper sensory input to the sacral and cortical micturition centres [64]. The combination of the failure to trigger the detrusor reflex and the urethral relaxation reflex results in detrusor underactivity and a nonrelaxing urethral sphincter which manifests as a voiding dysfunction. Normal urinary sensation is lost, and such patients depend on substitute sensations (i.e., fullness in the abdomen, tension in the pelvic region, a vague feeling of discomfort), which they gradually learn to associated with a full bladder [65]. The patients have to void at regular intervals and learn to stimulate the intrinsic neuromuscular mechanism by straining and other manipulations that allow them to void

by relaxing the voluntary sphincter [66]. However, voiding by straining is not a physiologic voiding method, and depends on the patient's abdominal muscle effort. It may compensate incompletely for the detrusor acontractility and urethral resistance [65]. Dysuria, chronic urinary retention, overflow incontinence, and frequent urinary tract infection are the common symptoms accompanying a voiding dysfunction. Some of the patients also need clean, intermittent self-catheterization [65].

In addition to impaired urinary bladder sensation and difficulty in micturition, urinary incontinence is also a common complaint of patients who undergo radical hysterectomy. A deficit in bladder neck support and the partial loss of alpha-adrenergic tone in the proximal urethra may result from the radical resection of the upper vagina and parametrium if there is damage to part or the pelvic plexus, resulting in a transaction of urethral sympathetic innervations [66]. Axelsen etal recently reported that the urethral sphincter mechanism plays a role in the pathophysiology, rather than the mobility of the bladder neck [67]. Bladder neck incompetence and low urethral closure pressure contribute to overflow incompetence and low urethral closure pressure contribute to overflow incontinence or urinary stress incontinence, which are exacerbated when combined with poor bladder compliance. The reduction in bladder compliance on filling may be due to a loss of sympathetic beta-adrenergic innervations with its inhibition and relaxation of the detrusor muscle [63]. Chronic urinary tract infection may also cause decreased bladder compliance [68].

Functional changes to the bladder and urethra after radical hysterectomy are multi-factorial. Anatomical changes and irreversible visceral damage after extensive dissections, as well as neurological alterations, may account for the resulting lower urinary tract dysfunction [69]. In other cases, pre-existing abnormal urinary tract function may worsen after radical hysterectomy [70]. Lin et al reported that up to 83% of patients with cervical cancer had abnormal urodynamic findings before radical hysterectomy [71]. Chen et al noted that the rate of detrusor instability before treatment in women with cervical carcinoma was higher than that of a control group with carcinoma-in-situ (CIN 3) (37.5% vs. 14.8%, $p < 0.05$) [72].

Urodynamic studies have made these functional disorders quantifiable. In 1949, Halter and Richter reported cystometric studies demonstrating a hypertonic bladder with reduced capacity in the early postoperative period. These findings have been confirmed by a number of authors [57, 64, &69]. Immediate postoperative hypertonic and poorly compliant bladder is believed to be related to perivesical adhesions and intrinsic myogenic tone change [73]. Overdistention in the early postoperative period should be avoided, for not only does it precipitate bladder atony but evidence also suggests that it may cause urinary fistulas [74].

Kuoet at performed urodynamic studies on 47 patients with cervical cancer who underwent radical hysterectomy and bilateral lymphadenectomy to assess the functional alteration of the bladder and urethra at different stages [69]. All patients developed hypertonic, poorly compliant bladder and reduction in urethral closure pressure before

bladder training was begun. After 3 to 6 months, 3 different types of cystometric change were noted. Twenty-five patients (53.2%) had hypotonic and acontractile bladders with enlarged capacity while 15 (31.9%) had better compliant bladder with normal tone but still had difficulty emptying the bladder. The remaining 7 patients (14.9%) maintained the immediate postoperative hypertonic state. Reduction of maximal urethral closure pressure was noted in 29 (61.8%) patients, remained unchanged in 13 (27.6%) and increased in 5 (10.6%). 43 patients had diminished or absent sensation of bladder fullness, and all except one complained of dysuria. The different urethral pressure changes and different cystometric changes documented in this study may reflect a more complicated mechanism after complete or incomplete denervation of autonomic nerves that control the lower urinary tract[69].

Generally, acute voiding symptoms disappeared within 6 to 12 months after radical hysterectomy [73]. However some authors have reported that 20% to 50% of similar patients have persistent urinary symptoms, mainly urinary incontinence, impaired bladder sensation, urinary tract infections, and straining to void [59]. Improvement of clinical symptoms alone was an unreliable indicator of improved function, since lower urinary tract dysfunction may be relieved by compensatory factors such as substitute sensations, abdominal straining, voiding technique, and the condition of the bladder outlet [73]. Such patients are permanently susceptible to decompensation, especially if placed in circumstances that promote bladder overdistention[74].

Dwyer et al investigated long-term symptomatic and urodynamic changes occurring in women with established urinary dysfunction after radical hysterectomy [75]. The only symptom that showed significant improvement over time was impaired sensation. There was no significant change in any of the urodynamic parameters between the initial and follow-up assessments. According to the study results, there was little improvement over time in urinary dysfunction following radical hysterectomy [75].

Methods for Minimizing Urinary Bladder Morbidity

Radicality is closely related to postoperative morbidity. Zullo et al recently noted that the extent of vaginal resection was more strongly associated with bladder dysfunction than was the extent of the lateral parametrial resection [76]. Lower-risk patients, i.e., those with smaller-volume tumors and in the early stages might benefit from modified radical hysterectomy (class II) which have been shown to cause less voiding dysfunction without compromising disease-free survival [77]. The type of the procedure may be different on each side of the cervix if tumor growth is asymmetrical [52]

Direct, nerve-sparing radical hysterectomy is a technique that spares the pelvic autonomic nerves without compromising radicality, providing another approach to improving quality of life and reducing bladder and bowel morbidity [77]. A number of studies have shown such surgical technique to be feasible with satisfactory recovery of voiding function [78, 79]. Raspagliesi et al reported that the type III nerve-sparing radical hysterectomy seems to be comparable to type II radical hysterectomy and superior to type III radical hysterectomy in terms of reducing early bladder dysfunction [80]. Todo et al

assessed postsurgical bladder function, 22 patients treated for cervical cancer with nerve-sparing radical hysterectomy by urodynamic study. There was no significant difference in compliance, maximal flow rate, and residual urine volume before the operation and at 12 months after the operation. This surgical technique is thought to be effective for preservation of bladder function [81].

The technique of sparing the pelvic autonomic nerves during radical hysterectomy for early stage cervical cancer and clinical stage II endometrial cancer is comparable to the conventional method in terms of perioperative complications and morbidity, but is more favorable in terms of return of bladder function [82].

Ditto A et al, compared class III nerve sparing radical hysterectomy (NSRH) compared with standard radical hysterectomy (RH) in cervical cancer (CC) in the context of multimodal therapies to evaluate disease-free survival, overall survival, local recurrence rate, and morbidities, in which total of 496 patients were enrolled and the median follow up was 93 months (42 and 159 months for the NSRH and RH groups, respectively) [89]. The oncologic results were comparable between NSRH and conventional class III RH in the context of two multimodal treatments. Bladder function and postoperative complications rate are improved by nerve sparing technique. The nerve sparing technique should be considered in all carcinoma cervix patients addressed to radical hysterectomy because it improves functional outcome and preserves radicality without compromising overall survival [83]. A recent meta-analysis demonstrated lower

incidence of LUTD following nerve sparing radical hysterectomy compared to conventional radical hysterectomy [84].

However, there are no standardized techniques for the nerve sparing approach. Urinary functional outcome varies according to the technique used and the different approaches to clear the ligament and the lymph nodes from the nerves. Laparoscopic and robotic approaches appear to facilitate the preservation of pelvic nerves by allowing a fine and precise dissection in a magnified operative field and are associated with better functional outcomes [85].

Unilateral nerve sparing approach is also associated with lower incidence of LUTD demonstrating that unilateral nerve injury could be partially compensated by the nervous supply from the other side [86].

In addition, there are different neural injuries with different prognosis. The injury could be a temporary blockade of signal transmission without axon lesion (neurapraxia) with functional disorders resolving hours to weeks following surgery. It could be a transection of the axon with intact nerve sheets (axonotmesis) allowing regeneration at the site of the injury and distal to the injury with a nerve growth velocity varying from 0.25mm/day to 4mm/day. A complete transection of the nerves including their sheets is also possible with no potential to regenerate [87]. The mechanism is even more complex because injury of vascular supply to these nerves is as much important as injuries to the nerves themselves [88]. The combination of stretch and ischemia makes the nerve more

vulnerable to injury. It appears that to obtain optimal functional results avoiding nerve handling in nerve sparing approach should be mandatory [88].

Finally, nerve injury is not the only mechanism responsible for LUTD after radical hysterectomy as demonstrated by the higher incidence of LUTD following type 4 nerve-sparing radical hysterectomy. Direct surgical injury to the bladder wall, lymph stasis, interruption of the blood supply, and fibrosis of the urethra also play a role [89].

A laparoscopic approach to reduce immediate postoperative morbidity has been an attractive prospect to surgeons in recent years [90].

Laparoscopic Radical Hysterectomy

Total laparoscopic radical hysterectomy for cervical carcinoma is an accepted modality not only because of its technical feasibility but also because of its oncologic outcomes [91]. However, bladder dysfunction after laparoscopic radical hysterectomy was actually increased without nerve sparing. Nerve-sparing techniques have been established to maintain postoperative quality of life. The hypogastric nerves are regarded as the anatomical landmark to accomplish total laparoscopic nerve-sparing radical hysterectomy. Bladder function following conventional total laparoscopic nerve-sparing radical hysterectomy is maintained. However, in some cases the radicality of this procedure is insufficient. Therefore, its applicability is expected to be limited to early stage cervical carcinoma [92]. Greater radicality is necessary for intermediate and advanced stage cervical carcinoma and may not permit nerve sparing in such cases[92].

Types of total laparoscopic radical nerve sparing hysterectomy [93]

1. Conventional nerve-sparing technique – Both sides hypogastric nerve and pelvic nerve plexus was preserved completely
2. Radical nerve-sparing technique – Both sides hypogastric nerves were sacrificed and pelvic splanchnic nerves were partially preserved
3. Non-nerve-sparing technique – Both sides hypogastric nerve and pelvic splanchnic nerves were completely sacrificed

The sensory nerve is distributed predominantly at the lower (dorsal) half of the pelvic nerve networks. The motor nerve is distributed predominantly at the upper (ventral) half of the pelvic nerve networks. The first sensation of bladder filling is weak and inconsistent, and is possibly dependent on cortical fluctuation. Impulses related to the first desire to void traverse through the pelvic nerves, and impulses for the sensation of a full bladder (strong desire to void) traverse through the pudendal nerves [94]. Therefore, first desire to void as the most effective parameter to evaluate bladder sensory function after the nerve-sparing procedure.

The sensory functions of conventional and radical nerve-sparing technique are statistically equivalent, and the sensory function of non-nerve-sparing technique is significantly lower than that of conventional and radical nerve-sparing technique. In contrast, the motor nerve is distributed predominantly at the upper (ventral) half of the pelvic nerve networks. Therefore, the motor function of conventional nerve-sparing technique is significantly more preserved compared to that of radical nerve-sparing and

non-nerve-sparing technique [93]. The motor functions of radical nerve-sparing and non-nerve-sparing technique are damaged similarly; they show no mutually significant difference. The various types of total laparoscopic nerve-sparing radical hysterectomies are technically feasible, and they can be tailored to the stage of cervical cancer [93].

Robotic Radical Hysterectomy

The clinical impact of robotic surgery in gynecologic field is growing widely, as suggested by a recent consensus statement made by the Society of Gynecologic Oncology in 2012 [95]. The society has stated that the current evidence supports at least an equivalence of robotic surgery and laparoscopy in many perioperative outcomes. However, there is a lack of disease specific oncologic outcomes and the cost is still a potential barrier to the widespread acceptance of robotic surgery [96]. Nevertheless, the number of robotic surgeries is growing and the implementation of robotic surgery in gynecologic oncology has made a dramatic change in the proportion of minimally invasive surgery, ranging from 3.3% to 43.5%, as shown in a study by Hoekstra, et al [97]. A recent meta-analysis indicated that comparison of robotic and conventional laparoscopic surgery was not feasible due to insufficiency in studies that assessed proper 'radical' hysterectomy solely for cervical cancer. Surgical outcomes of RRH and pelvic lymphadenectomy were comparable to that of laparoscopic approach, with favorable outcomes in regards to intraoperative blood loss and postoperative complications [98].

Therapy to Facilitate Urine Storage/Bladder Filling: [3]

Bladder related (inhibiting bladder contractility, decreasing sensory input, and/or increasing bladder capacity)

I. Behavioral therapy including any or all of the following:

1. Education
2. Bladder training
3. Timed bladder emptying or prompted voiding
4. Fluid restriction
5. Pelvic floor physiotherapy ± biofeedback

II. Pharmacologic therapy (oral, intravesical, intradetrusor)

1. Anti-cholinergic agents
2. Drugs with mixed actions
3. Calcium antagonists
4. Potassium channel openers
5. Prostaglandin inhibitors
6. β-Adrenergic agonists
7. α-Adrenergic antagonists
8. Tricyclic antidepressants; serotonin and norepinephrine reuptake inhibitors
9. Dimethyl sulfoxide (DMSO)
10. Polysynaptic inhibitors
11. Capsaicin, resiniferatoxin, and like agents

12. Botulinum toxin

III. Bladder overdistention

IV. Electrical stimulation and neuromodulation

V. Acupuncture and electroacupuncture

VI. Interruption of innervation

1. Very central (subarachnoid block)
2. Less central (sacral rhizotomy, selective sacral rhizotomy)
3. Peripheral motor and/or sensory

VII. Augmentation cystoplasty (auto, bowel, tissue engineering)

Outlet Related (Increasing Outlet Resistance)

I. Behavioral therapy

1. Education
2. Bladder training
3. Timed bladder emptying or prompted voiding
4. Fluid restriction
5. Pelvic floor physiotherapy ± biofeedback

II. Electrical stimulation

III. Pharmacologic therapy

1. α-Adrenergic agonists

2. Tricyclic antidepressants; serotonin and norepinephrine reuptake inhibitors

3. β-Adrenergic antagonists, agonists

IV. Vaginal and perineal occlusive and or supportive devices; urethral plugs

V. Nonsurgical periurethral bulking

VI. Collagen, synthetics, cell transfer (tissue engineering)

VII. Vesicourethral suspension ± prolapse repair (female)

VIII. Sling procedures ± proposal repair (female)

IX. Closure or compression of the bladder outlet (balloons, surgical closure)

X. "Sling/Tape" procedure (male)

XI. Artificial urinary sphincter

XII. Bladder outlet reconstruction

XIII. Myoplasty (muscle transposition)

XIV. Circumventing the problem

1. Absorbent products
2. External collecting devices
3. Anti-diuretic hormone like agents
4. Short acting diuretics
5. Intermittent catheterization
6. Continuous catheterization
7. Urinary diversion

Therapy to Facilitate Bladder Emptying/Voiding

Bladder related (increasing intravesical pressure or facilitating bladder contractility)

I. External compression, Valsalva

II. Promotion or initiating of reflex contraction

    1. Trigger zones or maneuvers
    2. Bladder "training"; tidal drainage

III. Pharmacologic therapy (oral, intravesical)

    1. Parasympathomimetic agents
    2. Prostaglandins
    3. Blockers of inhibition
        i. α-Adrenergic antagonists
        ii. Opioid antagonists

IV. Electrical stimulation

    1. Directly to the bladder or spinal cord
    2. Directly to the nerve roots
    3. Intravesical (transurethral)
    4. Neuromodulation

V. Reduction cystoplasty

VI. Bladder myoplasty (muscle wrap)

VII. Tissue engineering

Outlet related (Decreasing outlet resistance)

I. At level of smooth sphincter

1. Pharmacologic therapy - α-Adrenergic antagonists, β-Andrenergic agonists
2. Botulinum toxin (injection)
3. Transurethral resection or incision
4. Y-V plasty

II. At level of striated sphincter

1. Behavioural therapy ± biofeedback
2. Pharmacologic therapy
    i. Benzodiazepines
    ii. Baclofen
    iii. Dantrolene
    iv. α-Adrenergic antagonists
    v. Botulinum toxin (injection)
3. Urethral stent
4. Pudendal nerve interruption

III. Circumventing the problem

1. Intermitted catheterization
2. Continuous catheterization
3. Urinary diversion (conduit) [3].

AIM

The aim of this study was to analyze subjectively and objectively the urinary bladder morbidity in terms of voiding dysfunction following type III radical hysterectomy for carcinoma uterine cervix.

METHODS AND MATERIAL

This study was conducted after obtaining permission from the Institutional Ethics Committee, and it was in accordance with the Declaration of Helsinki & Good Clinical Practice (GCP) guidelines. Patients with cervix or carcinoma endometrium were explained about the study purpose and procedures. Informed consent was obtained in the language understandable to the individual.

Study type - Prospective study

Study period - January 2013 to January 2015

Sample size - 51 patients

Study population – Women diagnosed as carcinoma cervix or carcinoma endometrium.

Inclusion Criteria:

1. Age: 18 to 70 years
2. Carcinoma cervix up to Stage II with or without preoperative therapy
3. Carcinoma endometrium with endocervical involvement
4. Patients who underwent type III radical hysterectomy with curative indent

Exclusion Criteria:

1. Patients with preoperative urinary symptomatology (urgency, straining, urinary incontinence, dysuria)
2. Abnormalities in preoperative uroflowmetry
3. Patients with previous lower urinary tract surgery
4. Patients not willing to give informed consent

5. Patients not willing to do uroflowmetry in 1, 3, and 6 month

Study procedure:

Individuals who fulfilled the inclusion and exclusion criteria were enrolled to enter this study. All the patients underwent a complete clinical examination, including per vaginal and digital rectal examination and relevant radiological including chest X-ray and CT/MRI of abdomen and pelvis and other investigations required for diagnosis and staging. Multidisciplinary evaluations were done as indicated. Pre-anesthesia fitness was taken for all patients before surgery. Relevant history, co-morbidities, previous treatment and family history were obtained. All the patients were staged according to FIGO staging system after histopathological proof of cancer by biopsies. Patients received treatment according to their cancer site and stage as per standard practice. Open Type III Radical hysterectomy was done with standard surgical oncological principles.

A baseline screening for urinary bladder function was done at the diagnosis of disease prior to start of preoperative treatment, if any before radical hysterectomy followed by postoperative evaluation of bladder function was done at 1^{st} month, 3^{rd} month and 6^{th} month with urinary symptomatology (straining, urinary incontinence, urgency, and dysuria) and uroflowmetry (voided volume, maximum flow rate & average flow rate) and post-voided residual volume with ultra-sonogram. The patients who presented with incontinence in the first visit were educated about and encouraged to perform pelvic floor exercises. Patients were followed up for subsequent management of urinary problems, if any.

Statistical Analysis:

The variables investigated included age, body mass index, primary cancer, clinical stage, neoadjuvant or adjuvant therapy, urinary symptomatology (straining, incontinence, urgency, dysuria), parameters of uroflowmetry (voided volume, maximum flow rate, average flow rate), and post-void residual urine and needed catheterization.

Standard statistical tests Fischer exact test for categorical values and Wilcoxon signed rank test for sequential continuous variable were used for analysis. All the data were expressed as mean ± SD or in percentage as needed. The differences were considered to be significant if p value < 0.05.

Uroflowmetry:

Uroflowmetry is a test that measures the total voided volume of urine over time. This test is useful in evaluating the function of the lower urinary tract. Uroflowmetry is non-invasive and inexpensive test. It is easy to perform and quickly provides data on both storage and voiding symptoms. The addition of a non-invasive post-void residual volume measurement by ultrasound adds to the value of the study.

These studies were conducted with as much privacy as possible, and patients were asked to void urine when they feel a normal desire. The patients were instructed to urinate in a special urinal equipped with a computer machine that records the uroflow parameters. The patients were asked to press a button shortly before beginning urination, and to press the button again after finishing.

Preparation:

Uroflowmetry is best performed with full bladder. The patients were instructed not to urinate for 2 hours prior to the test. The patients were instructed to increase the volume of fluids they drink so as to have plenty of urine for the test.

Risks:

As the test involves normal urination and no invasive procedure, patients did not experience any discomfort. There was no risk associated with this test.

Normal Valves in Uroflow:

The maximum flow rate (Q max) is volume dependent, only voided volume of atleast 150 ml. should be interpreted [99]. The maximum flow rate should always be documented together with the total voided volume and post-void residual volume with the following standard format: maximum flow rate, volume voided, and post-void residual volume.

Normal uroflow parameter in a young man is Q max greater than 15-20 ml/sec and less than 10 ml/sec is abnormal. These numbers decline with age by 1-2 ml/sec per 5 years. There is a decline in peak flow with age resulting in a maximum flow of 5.5 ml/sec at 80 years [100].

Normal uroflow parameters in womenQ max can be greater than 30 ml/sec, and less than 15 ml/sec is abnormal [101]. Maximum flow in women does not seem to be dependent upon age. Women have a very short urethra, minimal outlet resistance, and no prostate, and generally speaking the only factors influencing female uroflow are the

strength of the detrusor muscle and the urethral resistance and the degree of relaxation of the sphincter mechanism [101].

Normal voiding includes a detrusor muscle contraction, co-ordinate bladder outlet relaxation, low voiding pressure, a smooth, arc-shaped flow curve [102]. The flow pattern, that is, the shape of the flow tracing, can sometimes be used to make a presumptive diagnosis, although it cannot be used to make a definitive diagnosis. The normal flow pattern is a continuous, bell-shaped smooth curve with a rapidly increasing flow rate [102].

Abnormal Uroflowmetry:

An intermittent flow pattern is one that has one or several episodes of flow increasing or decreasing (or ceasing completely) and is commonly secondary to abdominal straining or external sphincter spasm (e.g., detrusor-sphincter dys-synergia). The typical obstructed flow pattern has a plateau-shaped curve with a prolonged flow time, sustained low flow rate, and increased time to Q max. Abnormal results indicate urinary bladder morbidity which may be abnormal detrusor muscle contraction or uncoordinated bladder outlet relaxation but cannot distinguish true obstruction from poor detrusor contractility [103]. Maximum flow rate < 15 ml/sec may indicate detrusor weakness or outlet obstruction or both [10].

Post-void Residual Urine:

At the end of the urination, immediately after uroflowmetry, transabdominal ultrasound was done and the bladder was scanned in two planes and the three diameters

were measured. The formula for post-void residual urine used was: multiplying the products of three diameters (height × width × depth) by 0.625, a corrective factor as the bladder only approaches a spherical shape when it is full was to be applied [104]. Ultrasound obviates the need for urinal catheterization with its risk of introducing infection. Post-void residual urine ≥ 50 ml is abnormal.

RESULTS

In this study 51 patients underwent Type III radical hysterectomy (including bilateral pelvic lymph node dissection) with preoperative and postoperative evaluation of voiding dysfunction symptoms, uroflow parameters and post-void residual urine at 1st, 3rd, and 6th month. Data were expressed either in percentage or mean ± standard deviation as appropriate.

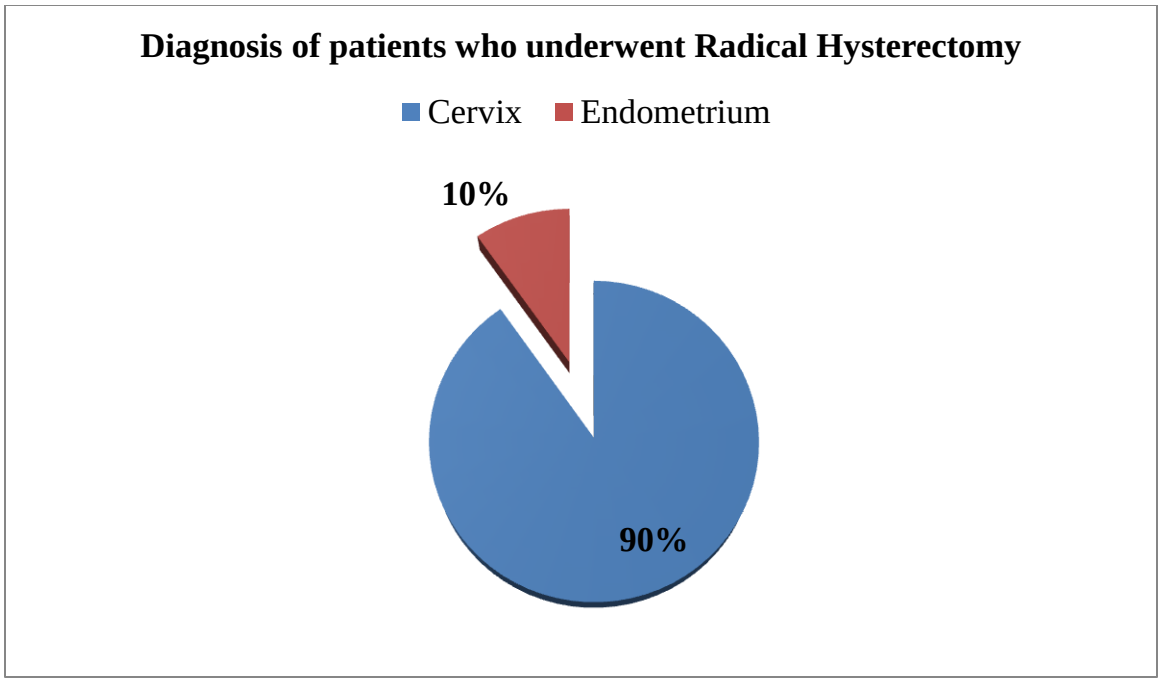
Table 1 - Baseline characteristics

Diagnosis	Patients	Age (years)	BMI
Carcinoma cervix	46 (90.2%)	49.04±9.51	20.11±2.03
Carcinoma endometrium with endocervical involvement	5 (9.8%)	56.20±7.63	25.48±1.34
Total	**51**	**49.75±9.52**	**20.64±2.54**

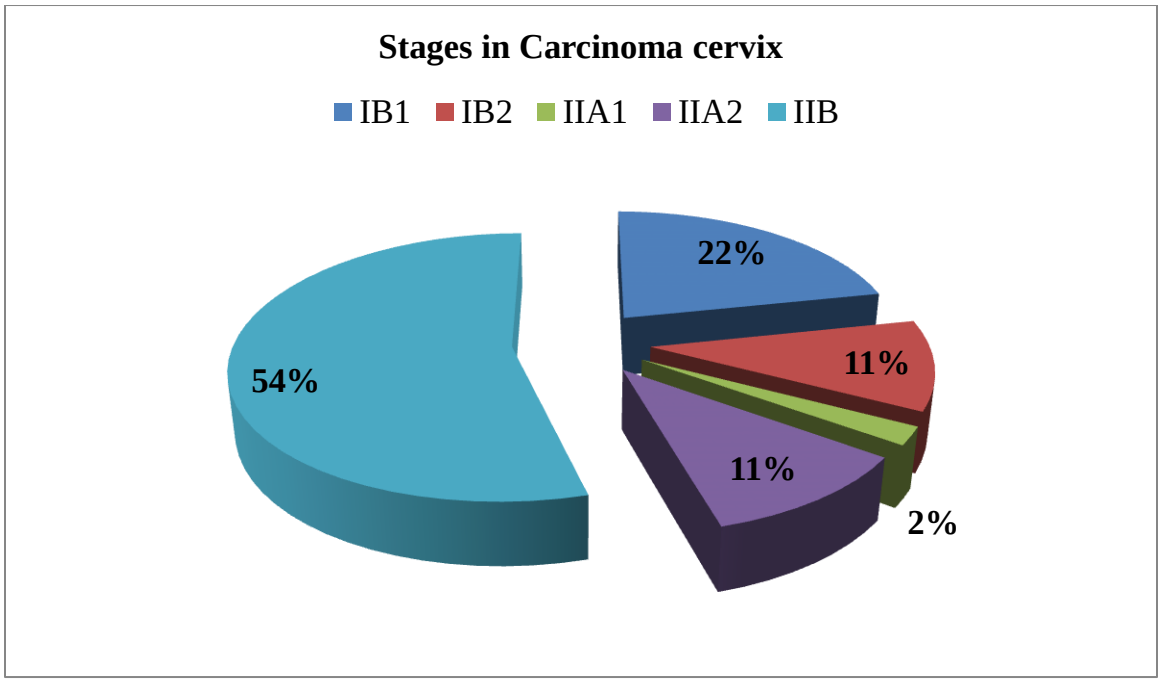
Age, BMI - Body mass Index expressed as mean ± standard deviation

Table 2 – Diagnosis and Stage

Diagnosis – Stage	Number	Total
Carcinoma cervix IB1	10	46
Carcinoma cervix IB2	5	
Carcinoma cervix IIA1	1	
Carcinoma cervix IIA2	5	
Carcinoma cervix IIB	25	
Carcinoma Endometrium with endocervical involvement (Stage II)	5	**5**



Out of 51 patients, 46 (90.2 %) patients had carcinoma cervix and 5 (9.8 %) patients had carcinoma endometrium [Table 1]. No significant differences in baseline characteristics - age and body mass index were observed.



In patients diagnosed as carcinoma cervix, 10, 5, 1, 5, and 25 patients had stage IB1, IB2, IIA1, IIA2, and II B respectively. Five patients diagnosed as carcinoma endometrium had endocervical involvement, clinically staged II [Table 2].

Table 3 - Modalities of Treatment in 51 patients

Modality	**RH**	**RH + RT**	**C + RH**	**C+RH+RT**	**RT + RH**	**CRT + RH**
Cervix I B1	9	1	0	0	0	0
Cervix I B2	0	0	2	2	0	1
Cervix II A1	0	1	0	0	0	0
Cervix II A2	0	0	1	0	2	2
Cervix II B	0	0	3	6	2	0
Endometrium II (Endocervical involvement)	2	3	0	0	0	3
Total	**11(21%)**	**5(10%)**	**6(12%)**	**8(16%)**	**4(8%)**	**17(33%)**

RH – Radical Hysterectomy,

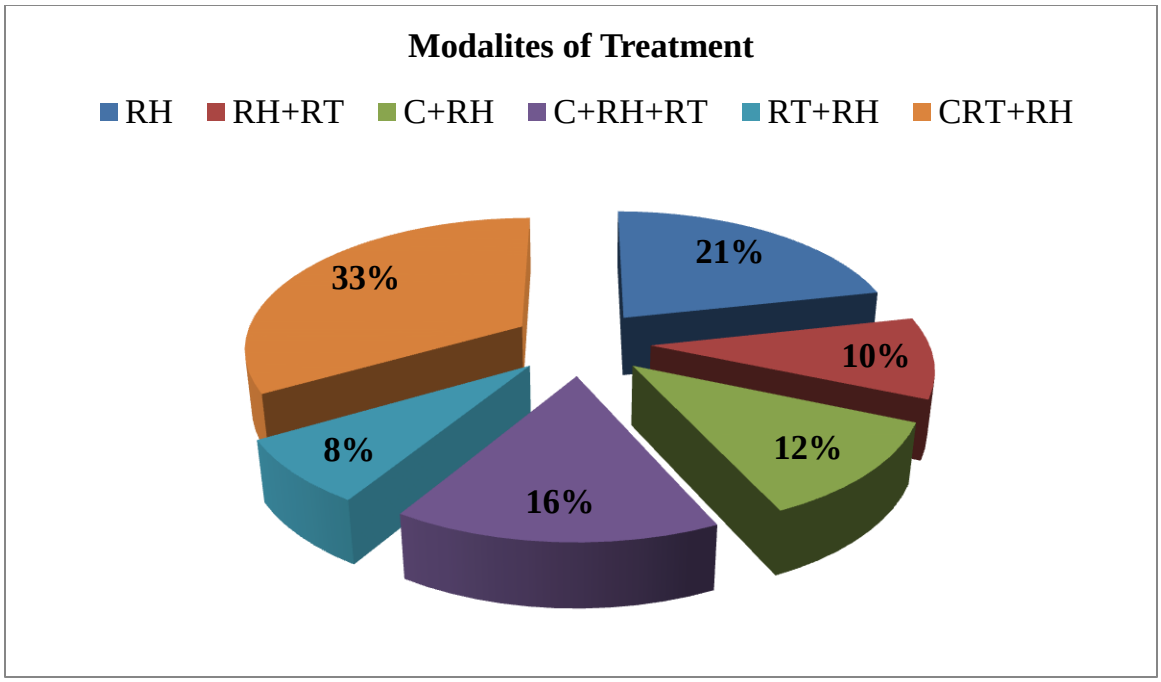
RH+RT –Radical Hysterectomy followed by adjuvant therapy,

C+RH – Preoperative Chemotherapy then Radical Hysterectomy,

C+RH+RT – Preoperative Chemotherapy then Radical Hysterectomy followed by adjuvant radiotherapy,

RT+RH – Preoperative radiotherapy then Radical Hysterectomy, and

CRT+RH – preoperative concurrent chemotherapy and radiotherapy then Radical Hysterectomy



. Out of 51 patients, 11 (21%) patients underwent radical hysterectomy and received no adjuvant therapy, 5 (10%) patients underwent radical hysterectomy and received adjuvant radiotherapy, 6 (12%) patients received preoperative chemotherapy followed by radical hysterectomy and received no adjuvant therapy, 8 (16%) patients received preoperative chemotherapy followed by radical hysterectomy and adjuvant radiotherapy, 4 (8%) patients received preoperative radiotherapy, 17 (33%) patients received preoperative concurrent chemoradiotherapy followed by radical hysterectomy and no adjuvant therapy (includes 3 patients who had received definitive concurrent chemoradiotherapy followed by radical hysterectomy for residual disease) [Table 3].

Out of 25 patients diagnosed as carcinoma cervix who underwent radical hysterectomy without preoperative radiotherapy, 10 patients received adjuvant radiation (8 patients received vaginal brachytherapy, and 2 patients received external beam

radiotherapy) [Table 4]. In 5 patients diagnosed as carcinoma endometrium, who underwent primary radical hysterectomy, 3 patients received adjuvant radiotherapy (2 patients received both external beam radiotherapy and vaginal brachytherapy, and 1 patient received vaginal brachytherapy alone) [Table 4].

Table 4 - Adjuvant Radiotherapy following Radical Hysterectomy

Carcinoma & Stage	Radical Hysterectomy n=30		Adjuvant Radiotherapy n=13			
	Primary	Preoperative Chemotherapy	EBRT +VB	EBRT	VB	TOTAL
Cervix I B1	10	0	0	1	0	1
Cervix I B2	0	4	0	0	2	2
Cervix II A1	1	0	0	1	0	1
Cervix II A2	0	1	0	0	0	0
Cervix II B	0	9	0	0	6	6
Endometrium II (Endocervical involvement)	5	0	2	0	1	3
Total	**16**	**14**	**2**	**2**	**9**	**13(43%)**

EBRT - External Beam Radiotherapy; VB - Vaginal Brachytherapy

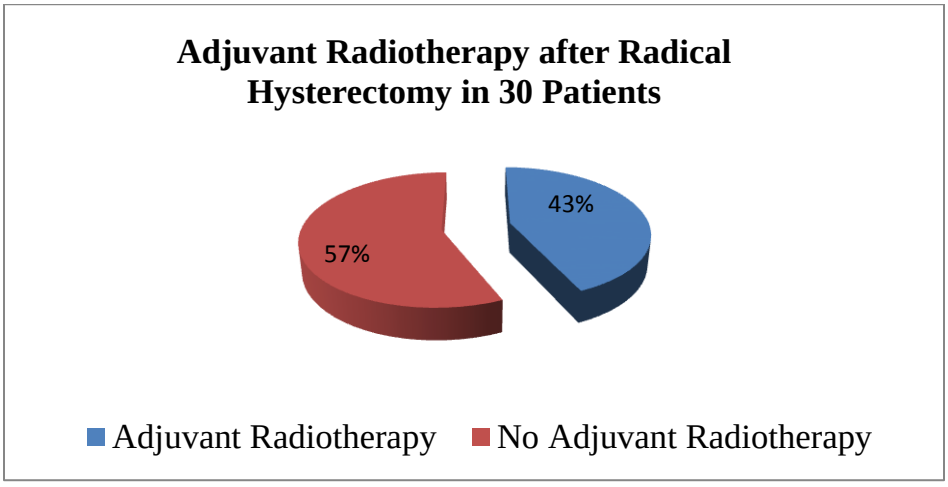


Table 5 – Complications and pathological parameters of radical hysterectomy

No	Parameters	Carcinoma Cervix (n=46)	Carcinoma Endometrium (n=5)	Total (n=51)
1	Preoperative radiotherapy ± Chemotherapy	21	0	**21**
2	No preoperative radiotherapy ± chemotherapy	25	5	**30**
	Adjuvant Radiotherapy	10	3	**13**
	EBRT(external beam radiotherapy)	2	1	**3**
	VB (vaginal brachytherapy)	8	0	**8**
	EBRT + VB	0	2	**2**
3	**Complications**			
	Operative mortality	0	0	**0**
	Wound infection	7	1	**8 (15.7%)**
	Preoperative radiotherapy (n=21)	5	0	**5 (23.8%)**
	No preoperative Radiotherapy (n=30)	2	1	**3 (10%)**

	Urinary bladder injury	0	0	**0**
	Ureteric injury	0	0	**0**
	Postoperative Urinary fistula	0	0	**0**
4	**Pathology**			
	Parametrium positive	0	0	**0**
	Vaginal margin positive	0	0	**0**
	Pelvic node positive	2	0	**2**
5	Recurrence within 6 months	0	1	**1**

Table 6 Pathological parameters of carcinoma endometrium

Carcinoma Endometrium	**Myometrial involvement (≥ half)**	**Grade**			**Cervical involvement**	
		1	**2**	**3**	**Stroma**	**Gland**
5	3	1	2	2	1	2

Out of 51 patients who underwent radical hysterectomy, there were no mortality and none of the patient had urinary bladder injury, ureteric injury, and postoperative urinary fistula. Overall wound infection rate was 15.7%. Wound infection rate in patients who had preoperative radiotherapy + radical hysterectomy was 23.8%, which was greater than in those who had no preoperative radiotherapy + radical hysterectomy (10%) [Table5].All patients who underwent radical hysterectomy had negative vaginal margin and none had microscopic/macroscopic parametrial involvement in definitive

histopathology. Only two patients had pelvic node positive disease. There were no loco-regional or distant recurrence at 6 months in patients with carcinoma cervix [Table 5].

In patients diagnosed with carcinoma endometrium only 1 patient showed pathological stage II and only one patient had vault recurrence at 6 month despite adjuvant vaginal brachytherapy [Table 5 &6].

Subjective Analysis - Symptoms

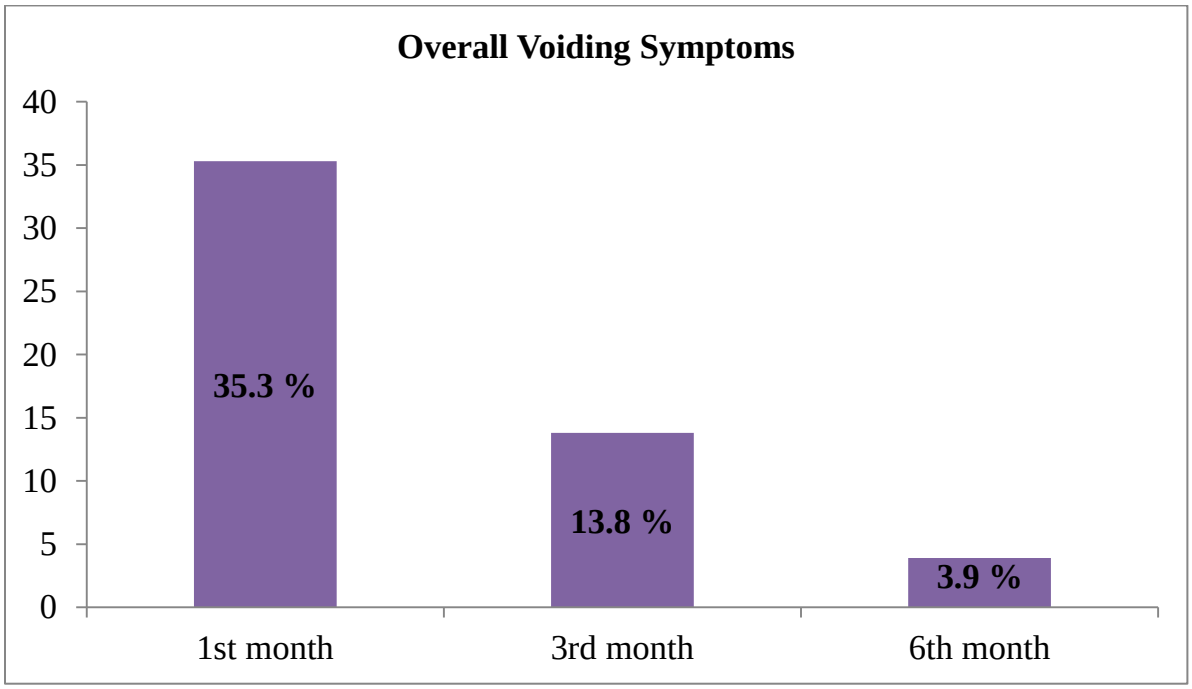
Table 7 - Voiding Symptoms in 51 patients

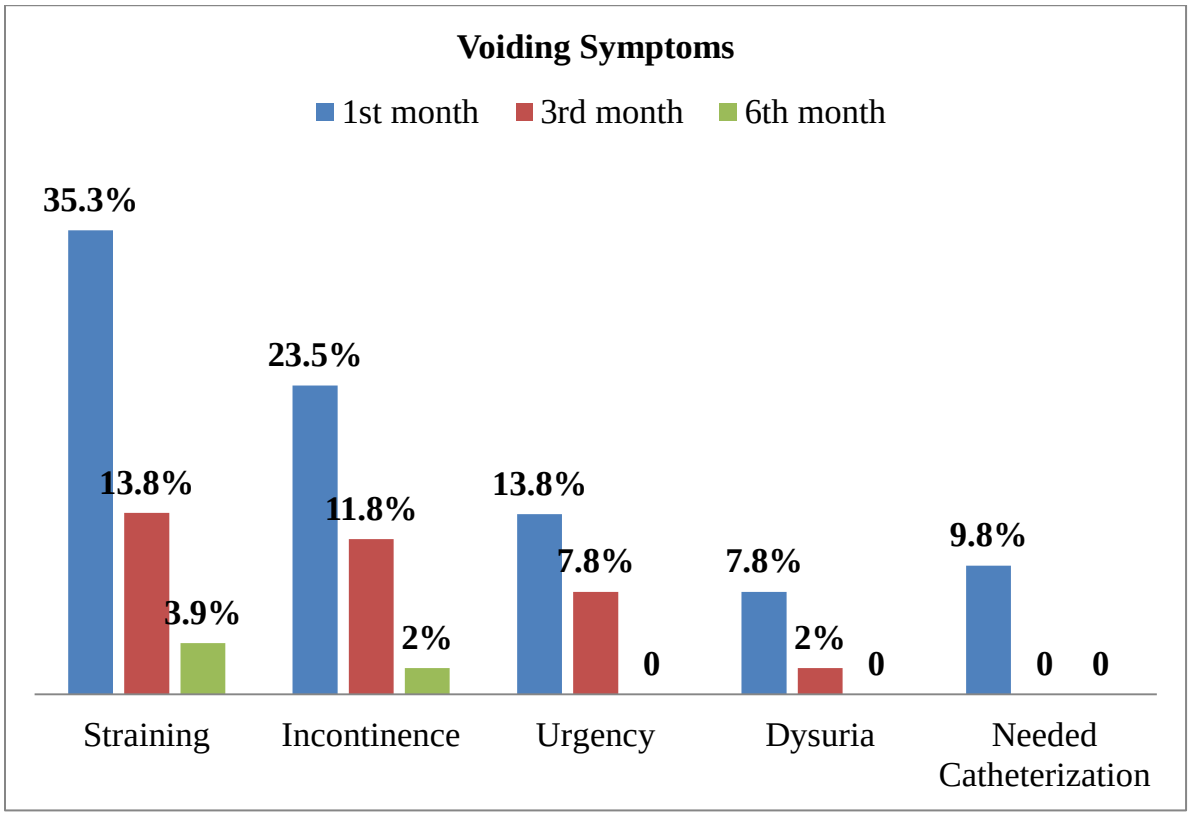
Symptoms	**1st month**	**3rd month**	**6th month**
Straining	18 (35.3 %)	7 (13.8 %)	2 (3.9 %)
Incontinence	12 (23.5 %)	6 (11.8 %)	1 (2.0 %)
Urgency	7 (13.8 %)	4 (7.8 %)	0
Dysuria	4 (7.8 %)	1(2.0%)	0
Needed Catheterization	5 (9.8 %)	0	0
Overall symptoms	**18 (35.3 %)**	**7 (13.8 %)**	**2 (3.9 %)**

The voiding symptoms presented by patients after radical hysterectomy were straining, urinary incontinence, urgency, and dysuria. All patients had normal bladder sensation at 1st, 3rd, and 6th month. The overall voiding dysfunction rate at 1st, 3rd, and 6th month after surgery were 35.3 %, 13.8 %, and 3.9% respectively.

The most common voiding symptom was straining at 1st, 3rd, and 6th month which manifested in 18 (35.3 %), 7 (13.8 %), and 2 (3.9%) patients respectively. Incontinence manifested in 12 (23.5 %), 6 (11.8%), and 1(2.0%) patients at 1st, 3rd and 6th month after

radical hysterectomy. Urgency manifested in 7 (13.7 %), and 4 (7.8 %) patients at 1st, and 3rd month respectively.





Dysuria manifested in 4 (7.8 %) and 1 (2.0 %) patients 1st, and 3rd month respectively. None had urgency and dysuria at 6 month after radical hysterectomy. 5 (9.8%) patients needed catheterization at 1st month only. No patient needed catheterization at 3rd and 6th month [Table 7].The rates of all the voiding symptoms decreased at 6 months when compared to 1st and 3rd month after radical hysterectomy [Table 7].

In 11 patients who underwent radical hysterectomy alone, none of the patients had incontinence, urgency, dysuria at any month. None needed catheterization at any month. Straining manifested only in 1 (9.1 %) patient at 1st month, none at 3rd and 6th month [Table 8].

In 5 patients who underwent radical hysterectomy followed by adjuvant radiotherapy, none of the patients had dysuria and needed catheterization at any month. None had voiding symptoms at 6th month. At 1st, and 3rd month only 1 patient (20%) manifested straining, incontinence, and urgency [Table 8].

In 6 patients who received preoperative chemotherapy followed by radical hysterectomy none had voiding symptom at 6th month. Straining and incontinence manifested in 2 patients (33.3%) at 1st month and in 1 (16.7 %) patient at 3rd month. Urgency manifested in 1 patient (16.7%) at 1st, and 3rd month. Dysuria manifested in 1 (16.7%) patient at 1st month only. Only 1 (16.7%) patient needed catheterization at 1st month [Table 8].

Table 8 - Voiding Symptoms

Modality	RH n=11	RH +RT n=5	C + RH n=6	C+RH+RT n=8	RT+RH n=4	CRT+RH n=17
Straining						
1st Month	1	1	2	3	1	10
3rd Month	0	1	1	1	0	4
6th Month	0	0	0	1	0	1
Incontinence						
1st Month	0	1	2	2	0	7
3rd Month	0	1	1	0	0	4
6th Month	0	0	0	0	0	1
Urgency						
1st Month	0	1	1	1	0	4
3rd Month	0	1	0	0	0	2
6th Month	0	0	0	0	0	0
Dysuria						
1st Month	0	0	1	1	0	2
3rd Month	0	0	0	0	0	1
6th Month	0	0	0	0	0	0
Needed Catheterization						
1st Month	0	0	1	1	1	3
3rd Month	0	0	0	0	0	0
6th Month	0	0	0	0	0	0

In 8 patients who received preoperative chemotherapy followed by radical hysterectomy and adjuvant radiotherapy, only one patient (12.5%) had urgency, dysuria, and needed catheterization at 1st month and none at 3rd and 6th month. Incontinence manifested in 2 patients (25%) at 1st month only and none at 3rd and 6th month. Straining manifested in 3 patients (37.5 %) in 1st month and 1 patient (12.5%) at 3rd and 6th month [Table 8].

In 4 patients who received preoperative radiotherapy followed by radical hysterectomy only one patient (25%) manifested straining at 1st month only, not at 3rdor 6th month. None manifested incontinence, urgency, and needed catheterization at any month. None had dysuria at any month [Table 8].

In 17 patients who received preoperative concurrent chemoradiotherapy 10 patients (58.8%), 4 patients (23.5%) and 1 patient (5.9%) had straining at 1st, 3rd and 6th month respectively. Incontinence manifested in 7(41.2%), 4 (23.5%), and 1 (5.9) patient at 1st, 3rd, and 6th month respectively. Urgency manifested in 4 (23.5%) and 2 (11.8%) patients at 1st, and 3rd month respectively and none at 6th month. Dysuria manifested in 2 (11.8%) and 1 (5.9%) patient at 1st, and 3rd month respectively and none at 6th month [Table 8].

The association of the voiding dysfunction symptom - straining between preoperative radiotherapy and no preoperative radiotherapy followed by radical hysterectomy was statistically significant only at 1st month but not at 3rd, and 6th month [Table 9].

Table 9 - Comparison of Voiding Symptoms

Preoperative Radiotherapy versus No Preoperative Radiotherapy

Symptoms	**RT ± C + RH**	**C ± RH ± RT**	**p Value**
1st month			
Straining	11	7	**0.0417**
Incontinence	7	5	0.1956
Urgency	4	3	0.4267
Dysuria	2	2	1
Catheterization	3	2	0.637
3rd month			
Straining	4	3	0.4267
Incontinence	4	2	0.2144
Urgency	2	2	1
Dysuria	1	0	0.4188
Catheterization	0	0	1
6th month			
Straining	1	1	1
Incontinence	1	0	1
Urgency	0	0	1
Dysuria	0	0	1
Catheterization	0	0	1

Table 10 - Comparison of Voiding Symptoms

Adjuvant Radiotherapy versus no Adjuvant Radiotherapy

Symptoms	**Adjuvant RT**	**No Adjuvant RT**	**p Value**
1st month			
Straining	4	3	0.6656
Incontinence	3	2	0.6278
Urgency	2	1	0.5645
Dysuria	1	1	1
Catheterization	1	1	1
3rd month			
Straining	2	1	0.5645
Incontinence	1	1	1
Urgency	1	1	1
Dysuria	0	0	1
Catheterization	0	0	1
6th month			
Straining	1	0	0.4333
Incontinence	0	0	1
Urgency	0	0	1
Dysuria	0	0	1
Catheterization	0	0	1

Table 11- Comparison of Voiding Symptoms

Preoperative Chemotherapy versus no Preoperative Chemotherapy

Symptoms	**RH**	**C+RH**	**p Value**
1st month			
Straining	1	2	0.5147
Incontinence	0	2	0.098
Urgency	0	1	0.3333
Dysuria	0	1	0.3333
Catheterization	0	1	0.3333
3rd month			
Straining	0	1	0.3529
Incontinence	0	1	0.3333
Urgency	0	0	1
Dysuria	0	0	1
Catheterization	0	0	1
6th month			
Straining	0	0	1
Incontinence	0	0	1
Urgency	0	0	1
Dysuria	0	0	1
Catheterization	0	0	1

The association of other voiding symptoms between preoperative radiotherapy and no preoperative radiotherapy followed by radical hysterectomy was not statistically significant at 1st, 3rd, and 6th month [Table 9].

The association of voiding symptoms between adjuvant radiotherapy and no adjuvant radiotherapy after radical hysterectomy was not statistically significant at 1st, 3rd, and 6th month [Table 10].

The association of voiding symptoms between preoperative chemotherapy and no preoperative chemotherapy was not statistically significant at 1st, 3rd, and 6th month [Table 11].

Table 12 - Comparison of chemotherapy and dysuria

Dysuria	**RH , RH + RT , RT + RH 11+5+4 = 19**	**C + RH , C+RH+RT , CRT + RH 6+8+17 = 31**	p value
1st month	0	4	0.2839
3rd month	0	1	1
6th month	0	0	1

Table 13 - Comparison radiotherapy and dysuria

Dysuria	**RH , C + RH 11+ 6= 17**	**RH + RT , RT + RH, C+RH+RT , CRT + RH 5+4+8+17 = 34**	p value
1st month	1	3	1
3rd month	0	1	1
6th month	0	0	1

The association of dysuria and chemotherapy was not statistically significant at 1st, 3rd, and 6th month [12].

The association of dysuria and radiotherapy was not statistically significant at 1st, 3rd, and 6th month [13].

Objective analysis - Uroflowmetry parameters

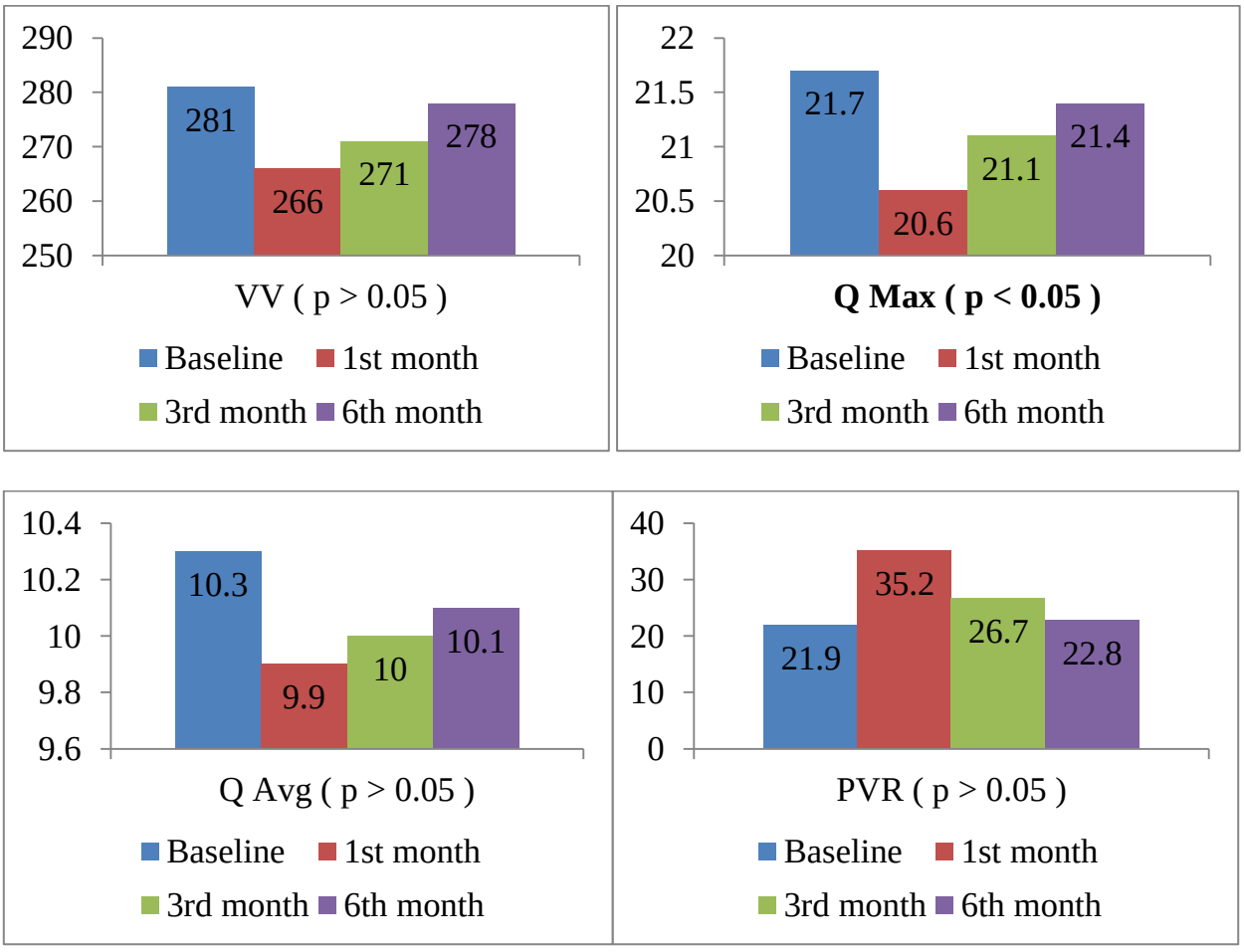
Table 14 – Uroflowmetry Parameters after Radical Hysterectomy in 51 Patients

Parameters	Preoperative	1st month (p value)	3rd month (p value)	6th month (p value)
VV	281±22	266±32(0.110)	271±33(0.188)	278±26(0.520)
Q Max	21.7±1.9	20.6±2.1**(0.011)**	21.1±2.1**(0.016)**	21.4±2.1**(0.045)**
Q Avg	10.3±0.4	9.9±0.7(0.171)	10.0±0.4(0.101)	10.1±0.4(0.082)
PVR	21.9±16.0	35.2±32.6(0.058)	26.7±22.3(0.171)	22.8±19.3(0.936)

All data expressed as mean ± standard deviation; p value – Wilcoxon signed rank test VV= Voided volume (mL); Q Max=Maximum flow rate (mL/sec); QAvg=Average flow rate (mL/sec); PVR=Post-void residual urine (mL).

In 51 patients who underwent radical hysterectomy, the preoperative voided volume, maximal flow rate, average flow rate and post-void residual urine were 281±22, 21.7±1.9, 10.3±0.4, and 21.9±16.0 respectively. At 1st month, 3rd month and 6th month after radical hysterectomy these parameters were 266 ± 32, 20.6 ± 2.1, 9.9 ± 0.7, &35.2 ± 32.6; 271 ± 33, 21.1 ± 2.1, 10.0 ± 0.4, & 26.7 ± 22.3; and 278 ± 26, 21.4 ± 2.1, 10.1 ± 0.4, & 22.8 ± 19.3 respectively [Table14].

Overall uroflow parameters after radical hysterectomy



In comparison of preoperative data with 1st, 3rd, and 6th month data, the mean value of voided volume, maximum flow rate and average flow rate decreased; and post-void residual urine increased [Table14].

In comparison of preoperative data with 1st, 3rd and 6th month data, only maximal flow rate revealed a statistically significant difference ($p < 0.05$). Other parameters voided volume, average flow rate and post-void residual urine were not significant at 1st, 3rd, and 6th month when compared with preoperative data [Table 14].

Table 15 - Comparison of uroflowmetry parameters between preoperative radiotherapy ± chemotherapy and no preoperative radiotherapy ± chemotherapy in radical hysterectomy

Parameters	Preoperative	1st month (p value)	3rd month (p value)	6th month (p value)
Group I: Preoperative Radiotherapy ± chemotherapy + RH n=21				
VV	286±22	270±32(0.289)	275±33(0.429)	288±27(0.569)
Q Max	21.5 ±2.0	19.9±1.8 **(0.012)**	20.5± 1.4 **(0.024)**	20.9±1.9 **(0.041)**
QAvg	10.4±0.4	9.9±0.7(0.408)	10.1±0.4(0.491)	10.2±0.4(0.471)
PVR	20.5±16.8	34.8±33.0(0.211)	27.4±22.6(0.171)	23.6±18.4(0.592)
Group II: No Preoperative Radiotherapy ± chemotherapy + RH n=30				
VV	277±21	262±32(0.121)	270±33(0.512)	273±23(0.313)
Q Max	21.9 ± 1.9	21.1 ± 2.1(0.298)	21.5 ± 2.3(0.222)	21.7 ± 2.2(0.373)
QAvg	10.3±0.5	9.9±0.7(0.293)	10.0±0.5(0.143)	10.1±0.5(0.109)
PVR	22.8±15.7	35.5±32.8(0.144)	26.2±22.4(0.497)	22.3±20.2(0.737)

All data expressed as mean ± standard deviation

To study the effect of preoperative radiotherapy in radical hysterectomy, the study population was divided into two groups namely group I with patients who underwent radical hysterectomy after preoperative radiotherapy with / without chemotherapy, and the group II with patients who underwent radical hysterectomy with/without preoperative chemotherapy but without preoperative radiotherapy. It was found that the maximum flow rate was the only parameter which was significant in Group I at 1st, 3rd, and 6thmonth

when compared with preoperative data. Other parameters voided volume, average flow rate and post-void residual urine were not significant at 1st, 3rd, and 6th month when compared with preoperative data [Table 15].

Table 16 - Comparison of uroflowmetry parameters between adjuvant radiotherapy and no adjuvant radiotherapy in radical hysterectomy (group II- no preoperative radiotherapy ± chemotherapy)

Parameters	Preoperative	1st month (p value)	3rd month (p value)	6th month (p value)
Group II: No Preoperative Radiotherapy ± chemotherapy + RH A: Adjuvant Radiotherapy n=13				
VV	276±20	268±36(0.808)	270±35(0.861)	270±25(0.718)
Q Max	22.0 ± 1.8	20.8 ±1.7(0.054)	21.3 ± 2.1**(0.017)**	21.6 ± 2.2(0.052)
QAvg	10.3±0.6	9.8±0.8(0.147)	9.9±0.5(0.181)	10.1±0.4(0.166)
PVR	25.0±14.9	31.9±31.0(0.608)	24.2±21.4 (0.887)	24.2±22.3(0.774)
Group II: No Preoperative Radiotherapy ± chemotherapy + RH B:No Adjuvant Radiotherapy n=17				
VV	278±23	258±28(0.090)	269±32(0.428)	274±22(0.196)
Q Max	21.9±2.0	21.4 ± 2.3(0.537)	21.7 ± 2.6 (0.600)	21.7 ± 2.2(0.775)
QAvg	10.3±0.4	9.9±0.6(0.942)	10.1±0.5(0.505)	10.1±0.5(0.404)
PVR	21.2±16.5	38.2±34.8(0.171)	27.6±23.7(0.260)	20.8±19.0(0.858)

All data expressed as mean ± standard deviation

To compare the effect of adjuvant radiotherapy, the Group II was sub-divided into Group A (received adjuvant radiotherapy) and Group B (received no adjuvant radiotherapy). In Group A, only the parameter maximum flow rate was found significant in 3rd month and not in 1st and 6th month when compared to preoperative data. Other parameters voided volume; average flow rate; and post-void residual urine were not significant in 1st, 3rd and 6th months when compared to preoperative data [Table 16].

Table 17 - Comparison of uroflowmetry parameters between no preoperative chemotherapy and preoperative chemotherapy followed by radical hysterectomy and no adjuvant radiotherapy

Parameters	Preoperative	1st month (p value)	3rd month (p value)	6th month (p value)
Radical Hysterectomy (RH) + No Adjuvant Radiotherapy n=11				
VV	280±23	268±23(0.442)	281±25(0.464)	277±23(0.448)
Q Max	21.8±1.6	21.8 ±2.0(0.674)	21.7±2.1(0.596)	21.6±1.8(0.752)
QAvg	10.3±0.3	10.0±0.5(0.914)	10.1±0.4(0.532)	10.1±0.4(0.527)
PVR	21.7±17.5	57.3±34.5(0.283)	28.2±25.3(0.297)	21.4±21.3(0.916)
Preoperative Chemotherapy + RH + No Adjuvant Radiotherapy n=6				
VV	273±24	238±26(0.115)	248±37(0.072)	268±22(0.180)
Q Max	22±2.7	20.6±3(0.247)	21.6±3.5(0.757)	21.9±3.0(0.474)
QAvg	10.4±0.4	9.8±0.8(0.784)	10±0.6(0.917)	10.2±0.7(0.371)
PVR	20.8±16.3	40.0±38.5(0.416)	26.7±22.7(0.715)	20.0±15.5(0.785)

None of the uroflow parameters were found to be significant when compared preoperative chemotherapy and no preoperative chemotherapy followed by radical hysterectomy at 1st, 3rd, and 6th month [Table 17].

Comparison of subjective and objective analysis of urinary bladder morbidity

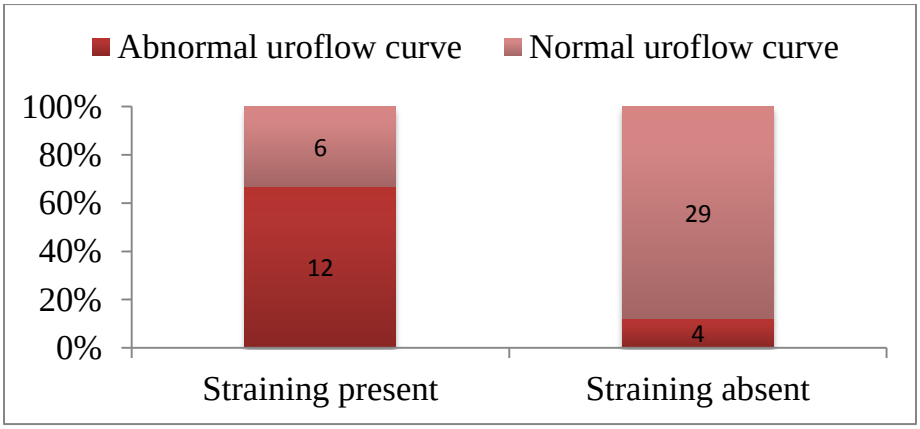
The association of straining and uroflow curve was considered to be statistically significant at 1st, 3rd, and 6th month [18].

Table 18- Comparison of Straining and Uroflow Curve

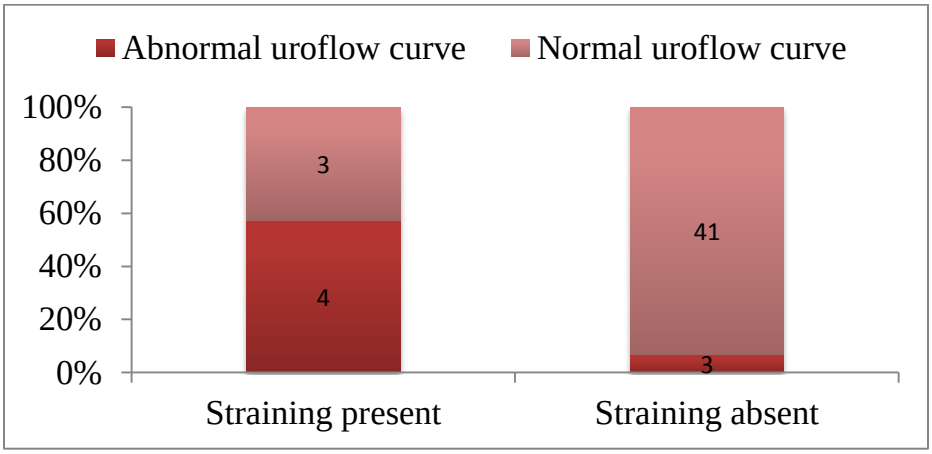
Straining	Uroflow curve		Total
	Abnormal	Normal	
1st month (p value= 0.0001)			
Present	12	6	**18**
Absent	4	29	**33**
Total	16	35	**51**
3rd month (p value = 0.0042)			
Present	4	3	**7**
Absent	3	41	**44**
Total	7	44	**51**
6thmonth (p value =0.0047)			
Present	2	0	**2**
Absent	2	47	**49**
Total	4	47	**51**

Comparison of Straining and Uroflow Curve

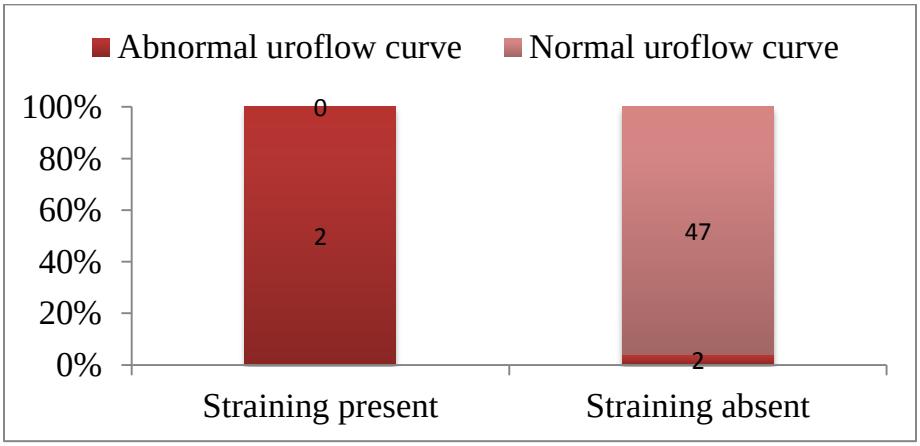
1st month



3rd month



6th month



The sensitivity, specificity, positive predictive value and negative predictive value of uroflow curve in identifying straining was 75%, 82.8%, 66.7%, & 87.9% ; 57.1%, 93.2%, 57.1%, & 93.2% ; and 50%, 95.9%, 100%, & 100% at 1st, 3rd, and 6th month respectively [Table18].

Table 19 - Comparison of Straining and Maximum Flow Rate

Straining	Maximum Flow Rate		Total
	Abnormal	Normal	
1st Month (p value =1)			
Present	0	18	**18**
Absent	0	33	**33**
Total	**0**	**51**	**51**
3rd Month (p value =1)			
Present	0	7	**7**
Absent	0	44	**44**
Total	**0**	**51**	**51**
6th Month (p value =1)			
Present	0	2	**2**
Absent	0	49	**49**
Total	**0**	**51**	**51**

The association between maximum flow rate and straining was not statistically significant at 1st, 3rd, and 6th month [Table19].

Table 20 - Comparison of Straining and Post void-residual urine

Straining	Post-void Residual Urine		Total
	Abnormal	Normal	
1st Month (p value 0.0002)			
Present	11	7	**18**
Absent	5	28	**33**
Total	**16**	**35**	**51**
3rd Month (p value 0.0276)			
Present	3	4	**7**
Absent	3	41	**44**
Total	**6**	**45**	**51**
6th Month (p value 0.1493)			
Present	1	1	**2**
Absent	3	46	**49**
Total	**4**	**47**	**51**

The association between post-void residual urine and voiding symptom straining was statistically significant at 1st month (p value 0.0022), and 3rd month (p value 0.0342) but was not statistically significant at 6th month (p value 0.3572) [Table 20].

At 1st month abnormal post-void residual urine had sensitivity, specificity, positive predictive value and negative predictive value of 68.8%, 80%, 61.1% and 84.8%

respectively in identifying voiding symptom straining [Table 20].At 3rd month abnormal post-void residual urine had sensitivity, specificity, positive predictive value and negative predictive value of 50%, 91.9%, 42.9% and 93.2% respectively in identifying voiding symptom straining [Table 20].

Table 21 - Comparison of Post void residual urine with Catheterization

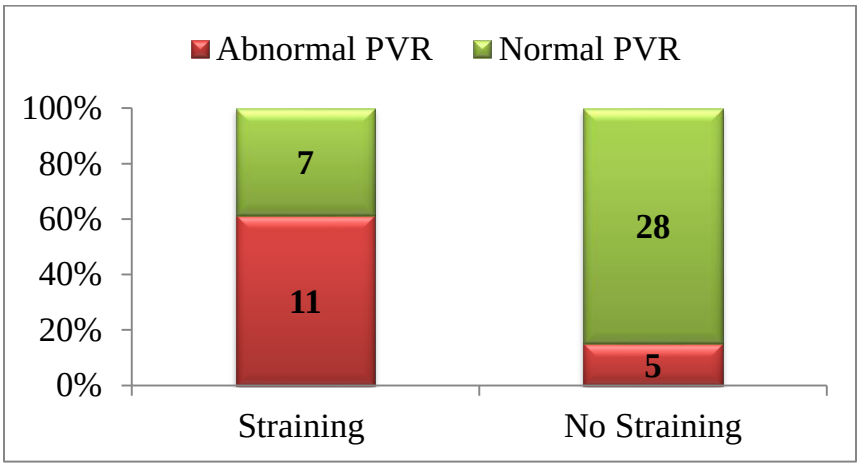
Catheterization	**Post-Void Residual Urine**		**Total**
	Abnormal	**Normal**	
1st month (p value= 0.0195)			
Needed	5	0	5
Not Needed	16	30	46
Total	21	30	51

The association between post-void residual urine and catheterization was statistically significant at 1st month (p value 0.0195), but not statistically significant at 3rd and 6th month [Table 21].

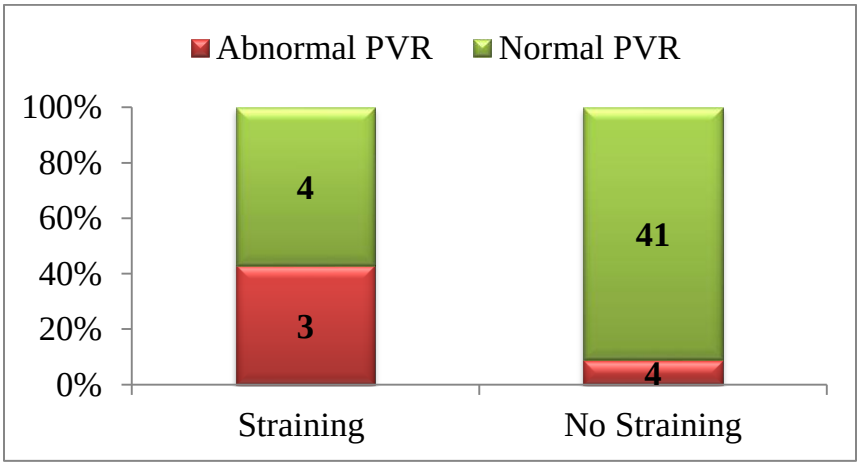
Post-void residual urine had sensitivity, specificity, positive predictive value and negative predictive value of 23.8%, 100%, 100% and 65.2% in identifying patients who needed catheterization at 1st month [Table 21].

Comparison of Straining and Post void-residual urine

1st month



3rd month



Comparison of Post void-residual urine and catheterization at 1st month

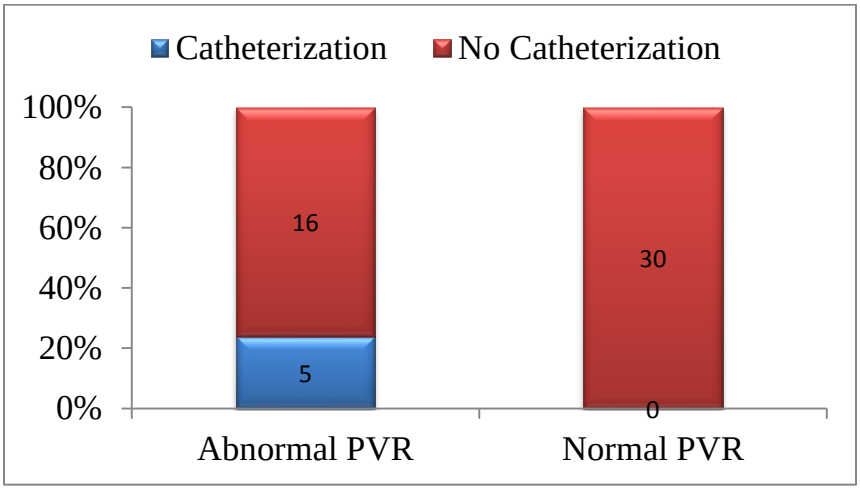


Table 22 - Comparison of Straining, Post-void residual urine and Catheterization

Comparison	Straining Present n = 18		Straining Absent n = 33		Total
Catheterization	Post-Void Residual Urine		Post-Void Residual Urine		
	Abnormal	Normal	Abnormal	Normal	
1st Month					
Needed	5	0	0	0	5
Not Needed	6	7	5	28	46
Total	11	7	5	28	51

In patients who had voiding symptom straining, the association between post-void residual urine and catheterization was not statistically significant at 1st month (p value = 0.1013). In patients who did not have voiding symptom straining, the association between post-void residual urine and catheterization was not statistically significant at 1st month (p value = 1).

Table 23 - Urine Culture in Patients with Dysuria

Month	Dysuria present	Urine Culture	
		Positive	Negative
1st	4	0	4
3rd	1	0	1

None had dysuria at 6th month. None of the patients with dysuria had positive urine culture at 1st and 3rd month [Table 23].

DISCUSSION

The incidence of urinary bladder dysfunction after radical hysterectomy for uterine cervical cancer is known to be between 70 % and 85 % [105, 106]. A few studies have suggested that the bladder dysfunction after radical hysterectomy may be transient and that bladder function may recover to baseline within 6 to 12 months [107]. In this study the rate of overall voiding symptom after type III radical hysterectomy was 35.3% at 1st month which decreased to 13.8% at 3rd month and 3.9% at 6th month. This showed improved voiding with time up to 6th month in this study. All patients had normal urinary bladder sensation after type III radical hysterectomy. In this study, the most common voiding symptom was straining, followed by incontinence, at 1st, 3rd, and 6th month. However, Sekido et al. reported postoperative urinary tract dysfunction that was sustained for more than 10 years [108].

In general, urinary bladder dysfunction after radical hysterectomy is known to be related to denervation of the autonomic nerve during surgical procedures. Chen et al. suggested that post-hysterectomy changes in lower urinary tract function may be related to the partial denervation of the pelvic autonomic nerves [71]. It has also been suggested by Zullo et al. that the most important factor for postoperative bladder dysfunction was the resection of vaginal and paravaginal tissue which supports the urinary bladder [75].

There was no operative mortality rate for type III radical hysterectomy in this study which was comparable to Meigs hysterectomy operative mortality rate [47]. There were no urinary bladder injury and no ureteric injury. No postoperative urinary fistula occurred, even though preoperative and postoperative radiotherapy was given. Overall wound infection rate was 15.7%. There were no recurrences in carcinoma cervix patients who underwent radical hysterectomy. Only 1 patient had vault recurrence in carcinoma endometrium, even though that patient received adjuvant vaginal brachytherapy.

Lin et al. reported that there was no significant difference in the results of urodynamic study between a radical hysterectomy only and a combination group that underwent radical hysterectomy and radiotherapy [109]. By contrast, Chauang and Kuo reported that patients who were treated by radical hysterectomy with radiotherapy had worse bladder morbidity. Thus, controversy remains about the effect of radiotherapy, and it is very difficult to solve this problem [110].

In this study, the association of straining between patients who received preoperative radiotherapy ± chemotherapy followed by radial hysterectomy and those who received no preoperative radiotherapy ± chemotherapy followed by radical hysterectomy showed a statistical significance at 1^{st} month only, but not at 3^{rd} and 6^{th} month. Other voiding symptoms didn't show a statistical significance at 1^{st}, 3^{rd}, and 6^{th} month.

The association of voiding symptoms between patients who received adjuvant radiotherapy after radical hysterectomy and no adjuvant therapy after radical hysterectomy didn't showed a statistical significance at 1st, 3rd, and 6th month. The association of voiding symptoms between patients who received preoperative chemotherapy followed by radical hysterectomy & no adjuvant therapy and no preoperative therapy followed by radical hysterectomy & no adjuvant therapy didn't showed a statistical significance at 1st, 3rd, and 6th month.

In this study, the decrease in maximum flow rate following radical hysterectomy showed a statistical significance at 1st, 3rd, and 6th month when compared with preoperative data, even though the actual mean value was not abnormal. Other parameters voided volume, average flow rate, and post-void residual urine were not statistically different at 1st, 3rd, and 6th month when compared with preoperative data.

It was observed that, patients who underwent radical hysterectomy with preoperative radiotherapy, had a decrease only in maximum flow rate only at 1st, 3rd, and 6th month with statistically significant difference when compared with patients who underwent radical hysterectomy without preoperative radiotherapy. Other parameters like voided volume, average flow rate and post void residual urine didn't showed a statistical significance at 1st, 3rd and 6th month. .

Comparing patients who had received adjuvant radiotherapy versus no adjuvant radiotherapy following radical hysterectomy, patients who received adjuvant radiotherapy had a statistical significance decrease in maximum flow rate at 3rdmonth only, as adjuvant radiotherapy was started 4 to 6 weeks after radical hysterectomy. Other parameters were not significant at any time. The results of this study support the theory that radiotherapy may result in the deterioration of postoperative bladder function and it may recover to baseline within 6thmonth.

Comparing preoperative chemotherapy and no preoperative chemotherapy in patients who underwent radical hysterectomy, none of the uroflow parameters showed a statistical significance at 1st, 3rd, and 6th month.

Even though the maximum flow rate was decreased at 6 month with statistically significance (p value 0.045), the mean value was not abnormal, and voiding symptoms improved at 6thmonth (3.9%) when compared to 1st month (35.3%) and 3rd month (13.8%), there was no statistical significant association between uroflow parameter maximum flow rate and straining symptom. In spite of this, uroflow curve was found be abnormal in symptomatic patients who had straining with statistical significance at 1st, 3rd, and 6th month.

Incontinence rate decreased from 23.5% to 2% at 6th month as pelvic floor exercises were reinforced to patients. Urgency completely disappeared at 6th month from 13.7% at 1st month and 7.8% at 3rd month. Dysuria manifested in 9.8% and 2% at 1st and 3rd month respectively and none at 6th month. None of the patients with dysuria had a positive urine culture. Dysuria never manifested in patients who were not exposed to chemotherapy in their treatment but unable to prove statistical significance. Only 9.8% needed catheterization at 1st month and none at 3rd and 6th month.

In this study, post-void residual urine after type III radical hysterectomy at 1st, 3rd, and 6th month was not significant when compared with preoperative data. However the association of post-void residual urine with voiding symptom straining showed statistical significance at 1st and 3rd month but not at 6th month. The association of post-void residual urine and the need of catheterization showed statistical significance at 1st month. But in patients with straining the association of post-void residual urine and the need of catheterization was not found to be statistically significant.

This study has clinical significance because few studies [111] have assessed the effect of radiotherapy preoperatively as well as postoperatively and chemotherapy by use of uroflowmetry and post-void residual urine. Considering that role of radiotherapy is inevitable for carcinoma cervix and endometrium, our final goal of treatment should be gaining not only good oncologic outcomes but also the prevention of lower urinary tract morbidity.

LIMITATIONS OF THE STUDY

1. The artificial situation of the urodynamic laboratory might produce a non-physiologic result.
2. The absence of a specific abnormality during urodynamic testing does not exclude its existence.
3. Only non-invasive uroflowmetry and measured of post-void residual urine by transabdominal ultrasound was used in this study, invasive urodynamic study such as cystometry and pressure flow studies were not used and measurement of post-void residual urine was not measured by catheterization.
4. Inconsistent reproducibility of test results in the same patient when repeated.
5. Wide range of physiologic values in normal, asymptomatic patients.
6. Only short-term (6 months) urinary bladder morbidity was assessed with small sample size (n=51) in our study.

CONCLUSION

In conclusion, type III radical hysterectomy gives rise to transient alteration in the neurophysiology of the lower urinary tract. Although most of these changes return to normal within a certain period of time (6-12 months), it is suggested to pay attention to voiding symptom of straining, and the uroflow parameter of maximum flow rate, especially in patients who receive radiotherapy. The uroflow curve and post-void residual urine in also require note in patients with voiding symptom straining.

This study showed that it may not always be necessary to use uroflowmetry values to evaluate the voiding dysfunction following radical hysterectomy, as there is wide range of physiologic values in asymptomatic patients and not all abnormalities in uroflowmetry are clinically significant. Post-void residual urine measured with non-invasive method of ultrasound may be sufficient to identify patients who will need intermittent catheterization. Further studies with a larger number of patients and long-term follow-up are required to explore this.

BIBLIOGRAPHY

1. Blaivas and Chancellor, 1995 a. Blaivas JG, Chancellor MB: Caudaequina and pelvic plexus injury .In: Chancellor MB, Blaivas JG, ed. Practical Neurourology, Boston : Butterworth-Heinemann; 1995:155-164.
2. McGuire, 1984. McGuire EJ: Clinical evaluation and treatment of neurogenic vesical dysfunction. In: Libertino JA, ed. International Perspectives in Urology, vol 11. Baltimore: Williams & Wilkins; 1984:43-56
3. Wein and Barrett, 1982. Wein AJ, Barrett DM: Etiologic possibilities for increased pelvic floor electromyographic activity during cystometry. J Urol 1982; 127:949-952.
4. deGroat and Booth, 1993. deGroat WC, Booth AM: Synaptic transmission in pelvic ganglia. In: Maggi CA, ed. The Autonomic Nervous System, vol 3, Nervous Control of the Urogenital System, London: Harwood Academic; 1993:291-347
5. Elbadawi and Schenk, 1966. Elbadawi A, Schenk EA: Dual innervation of the mammalian urinary bladder, a histochemical study of the distributions of cholinergic and adrenergic nerves. Am J Anat 1966; 119:405.
6. Donker et al., 1982. Donker PJ, Droes JP, Van Alder BM: Anatomy of the musculature and innovation of the bladder and urethra. In: Chisolm GO, Williams DI, ed. Scientific Foundations of Urology, Chicago: Year Book; 1982:404-441.

7. Tanagho, 1982. Tanagho EA: The ureterovesical junction: Anatomy and physiology. In: Chishold GD, Williams DI, ed. Scientific Foundation of Urology, Chicago: Year Book; 1982:295-404.
8. Chancellor et al., 1996. Chancellor MB, Rivas DA, Bourgois IM: Laplace's law and the risks and prevention of bladder rupture after enterocystoplasty and bladder autoaugmentation. NeurourolUrodyn 1996; 15:223.
9. Andersson, 1993. Andersson KE: Pharmacology of lower urinary tract smooth muscles and penile erectile tissues. Pharmacol Rev 1993; 45:253.
10. Thor et al., 1989. Thor KB, Morgan C, Nadelhaft I, et al: Organization of afferent and efferent pathways in the pudendal nerve of the female cat. J Comp Neurol 1989; 288:263.
11. Janig and Morrison, 1986. Janig W, Morrison JFB: Functional properties of spinal visceral afferents supplying abdominal and pelvic organs, with special emphasis on visceral nociception. Prog Brain Res 1986; 67:87.
12. Bosch and Groen, 1995. Bosch JL, Groen J: Sacral (S3) segmental nerve stimulation as a treatment for urge incontinence in patients with detrusor instability: Results of chronic electrical stimulation using an implantable neural prosthesis. J Urol 1995; 154:504
13. Barrington, 1921. Barrington FJF: The relation of the hindbrain to micturition. Brain 1921; 44:23.

14. Blok and Holstege, 1996. Blok BF, Holstege G: Neuronal control of micturition and its relation to the emotional motor system. Prog Brain Res 1996; 107:113.
15. Ohtake et al., 2004. Ohtake A, Ukai M, Hatanaka T, et al: In vitro and in vivo tissue selectivity profile of solifenacin succinate (YM905) for urinary bladder over salivary gland in rats. Eur J Pharmacol 2004; 492:243.
16. Valentino et al., 1995. Valentino RJ, Pavcovich LA, Hirata H: Evidence for corticotropin-releasing hormone projections from Barrington's nucleus to the periaqueductal gray and dorsal motor nucleus of the vagus in the rat. J Comp Neurol 1995; 363:402.
17. Sugaya et al., 2003. Sugaya K, Kadekawa K, Ikehara A, et al: Influence of hypertension on lower urinary tract symptoms in benign prostatic hyperplasia. Int J Urol 2003; 10:569.
18. Andersson, 2004. Andersson KE: Mechanisms of disease: Central nervous system involvement in overactive bladder syndrome. Nat ClinPractUrol 2004; 1:103-108.
19. Brading, 1999. Brading AF: The physiology of the mammalian outflow tract. ExpPhysiol 1999; 84:215-221.
20. Wein and Barrett, 1988. Wein AJ, Barrett DM: Voiding Function and Dysfunction—A Logical and Practical Approach, Chicago, Year Book Medical, 1988.

21. Zinner et al., 1983. Zinner NR, Sterling AM, Ritter R: Structure and forces of continence. In: Raz S, ed. Female Urology, Philadelphia: WB Saunders; 1983:33-41.

22. Andersson and Arner, 2004. Andersson K-E, Arner A: Urinary bladder contraction and relaxation: Physiology and pathophysiology. Physiol Rev 2004; 84:935-988.

23. Enhorning, 1961. Enhorning G: Simultaneous recording of intravesical and intraurethral pressure. ActaChirScand 1961; 276(Suppl):1-68.

24. Chancellor and Yoshimura, 2002. Chancellor MB, Yoshimura N: Physiology and pharmacology of the bladder and urethra.
In: Walsh PC, Retik AB, Vaughan Jr ED, Wein AJ, ed. Campbell's Urology, Philadelphia: Elsevier Science (Saunders); 2002:831-886.

25. Wein, 1981. Wein AJ: Classification of neurogenic voiding dysfunction. J Urol 1981; 125:605-609

26. Abrams P, Cardozo L, Fall M, Griffiths D, Rosier P, Ulmsten U, van Kerrebroeck P, Victor A, Wein A; Standardisation Sub-committee of the International Continence Society. The standardisation of terminology of lower urinary tract function: report from the Standardisation Sub-committee of the International Continence Society. NeurourolUrodyn. 2002; 21(2):167-78.

27. Jensen JK, Nielsen FR Jr, Ostergard DR. The role of patient history in the diagnosis of urinary incontinence. ObstetGynecol 1994; 83:904.

28. 7. Ward RM, Hampton BS, Blume JD, et al. The impact of multichannel urodynamics upon treatment recommendations for female urinary incontinence. IntUrogynecol J Pelvic Floor Dysfunct
29. Glazener CM, Lapitan MC. Urodynamic investigations for management of urinary incontinence in adults. Cochrane Database Syst Rev 2002; :CD003195
30. Sand PK, Hill RC, Ostergard DR. Supine urethroscopic and standing cystometry as screening methods for the detection of detrusor instability. ObstetGynecol 1987; 70:57.
31. Barnick, CG, Cardozo, DL, Benness, C. Use of routine videocystourethrography in the evaluation of female lower urinary tract dysfunction. NeurourolUrodyn 1989; 8:447.
32. Patravali N. Ambulatory urodynamic monitoring: are we wasting our time? J ObstetGynaecol 2007; 27:413
33. Bergman A, Bhatia NN. Uroflowmetry for predicting postoperative voiding difficulties in women with stress urinary incontinence. Br J ObstetGynaecol 1985; 92:835.
34. Haylen BT, Ashby D, Sutherst JR, et al. Maximum and average urine flow rates in normal male and female populations--the Liverpool nomograms. Br J Urol 1989; 64:30.
35. Nitti VW, Raz S. Obstruction following anti-incontinence procedures: diagnosis and treatment with transvaginalurethrolysis. J Urol 1994; 152:93.

36. Defreitas GA, Zimmern PE, Lemack GE, Shariat SF. Refining diagnosis of anatomic female bladder outlet obstruction: comparison of pressure-flow study parameters in clinically obstructed women with those of normal controls. Urology 2004; 64:675
37. Rosenzweig BA, Bhatia NN, Nelson AL. Dynamic urethral pressure profilometry pressure transmission ratio: what do the numbers really mean? ObstetGynecol 1991; 77:586.
38. Miklos JR, Sze EH, Karram MM. A critical appraisal of the methods of measuring leak-point pressures in women with stress incontinence. ObstetGynecol 1995; 86:349.
39. Vereecken RL. A critical view on the value of urodynamics in non-neurogenic incontinence in women. IntUrogynecol J Pelvic Floor Dysfunct 2000; 11:188.
40. 10. Maniam P, Goldman HB. Removal of transurethral catheter during urodynamics may unmask stress urinary incontinence. J Urol 2002; 167:2080.
41. 11. Lose, G, Thyssen, H. Reproducibility of cystometry and pressure-flow parameters in female patients. NeurourolUrodyn 1996; 15:302.
42. 12. Versi E. Discriminant analysis of urethral pressure profilometry data for the diagnosis of genuine stress incontinence. Br J ObstetGynaecol 1990; 97:251.
43. Baskett TF. Hysterectomy: evolution and trends. Best Pract Res ClinObstetGynaecol 2005; 19:295-305.

44. Holland CM, Shafi MI. Radical hysterectomy. Best Pract Res ClinObstetGynaecol 2005; 19:387-401.
45. Okabayashi H. Radical abdominal hysterectomy for cancer of the cervix uteri, modification of the takayama operation. SurgGynecolObstet1921; 33: 335-341.\
46. Mibayashi R **[**Results in the treatment of cervical cancer at the Kyoto University obstetrical and gynecological clinic].J JpnObstetGynecolSoc 14:471, 1962 471-2
47. Meigs JV. Carcinoma of the cervix—the Wertheim operation. SurgGynecolObstet 1944; **78:** 195–98.
48. Kobayashi T. Abdominal radical hysterectomy with pelvic lymphadenectomy for cancer of cervix. 2nd ed. Nanzando, Tokyo 1961; 178-187.
49. Michel Canis, Gerard Mage, Charles Chaperon, Arnaud Wattier, Jean LcPouly, and Maurice Antoine Bruhat. Laproscopic hysterectomy – A preliminary study. SurgEndosc(1993) 7;42-45.
50. Nezhat CR, Nezhat FR, Burrell MO, Ramirez CE, Welander C, Carrodeguas J, NezhatCH.Laparoscopic radical hysterectomy and laparoscopically assisted vaginal radical hysterectomy with pelvic and paraaortic node dissection.JGynecol Surg. 1993 Summer; 9(2):105-20.
51. Sert BM, Abeler VM. Robotic-assisted laparoscopic radical hysterectomy (Piver type III) with pelvic node dissection: casereport. Eur J GynaecolOncol 2006; 27: 531-3.

52. Querleu D, Morrow CP. Classification of radical hysterectomy. Lancet Oncol 2008; 9:297
53. Piver MS, Rutledge F, Smith JP. Five classes of extended hysterectomy for women with cervical cancer. ObstetGynecol 1974; 44:265.
54. Mota F, Vergote I, Trimbos JB, et al. Classification of radical hysterectomy adopted by the Gynecological Cancer Group of the European Organization for Research and Treatment of Cancer. Int J Gynecol Cancer 2008;18(5):1136-1138.
55. Raspagliesi F, Ditto A, Fontanelli R, et al. Nerve-sparing radical hysterectomy: a surgical technique for preserving the autonomic hypogastric nerve. GynecolOncol 2004; **93:** 307 14.
56. Trimbos JB, Maas CP, Deruiter MC, et al. A nerve-sparing radical hysterectomy: guidelines and feasibility in Western patients. Int J Gynecol Cancer 2001; **11:** 180–86.
57. Ralph G, Tamussino K, Lichtenegger W: Urological complications after radical abdominal hysterectomy for cervical cancer. BaillieresClinObstetGynaecol 1988; 2:943-952
58. Smith PH, Turnbull GA, Currie DW, Peel KR: The urological complications of Wertheim's hysterectomy. Br J Urol 1969; 41:685-688
59. Macasaet MA, Lu T, Nelson JH: Ureterovaginal fistula as a complication of radical pelvic surgery. Am J ObstetGynecol 1976; 124: 757-760

60. Twombly GH, Landers D: The innervation of the bladder with reference to radical hysterectomy. Am J ObstetGynecol 1956; 71:1291- 1300.
61. Elbadawi A, Schenk EA: A new theory of the innervation of bladder musculature. 3. Postganglionic synapses in uretero-vesico-urethral autonomic pathways. J Urol 1971; 105:372-374.
62. Forney JP: The effect of radical hysterectomy on bladder physiology. Am J ObstetGynecol 1980; 138:374-382
63. Ralph G, Winter R, Michelitsch L, Tamussino K: Radicality of parametrial resection and dysfunction of the lower urinary tract after radical hysterectomy. Eur J GynaecolOncol 1991; 12:27-30.
64. Seski JC, Diokno AC: Bladder dysfunction after radical abdominal hysterectomy. Am J ObstetGynecol 1977; 128:643-651.
65. Sekido N, Kawai K, Akaza H: Lower urinary tract dysfunction as persistent complication of radical hysterectomy. Int J Urol 1997; 4: 259-264
66. Low JA, Mauger GM, Carmichael JA: The effect of Wertheim hysterectomy upon bladder and urethral function. Am J ObstetGynecol 1981; 139:826-834
67. Axelsen SM, Bek KM, Petersen LK: Urodynamic and ultrasound characteristics of incontinence after radical hysterectomy. NeurourolUrodyn 2007 [Epub ahead of print].
68. Woodside JR, McGuire EJ: Detrusor hypertonicity as a late complication of a Wertheim hysterectomy. J Urol 1982; 127:1143-1145

69. Kuo HC, Tsai TC, Hsieh CY, Lee CJ: Voiding dysfunction after radical abdominal hysterectomy. Taiwan Yi XueHuiZaZhi 1985; 84: 1137-1146.

70. Lin LY, Wu JH, Yang CW, Sheu BC, Lin HH: Impact of radical hysterectomy for cervical cancer on urodynamic findings. IntUrogynecol J Pelvic Floor Dysfunct 2004; 15:418-421.

71. Lin HH, Yu HJ, Sheu BC, Huang SC: Importance of urodynamic study before radical hysterectomy for cervical cancer. GynecolOncol 2001; 81:270-272.

72. Chen GD, Lin LY, Wang PH, Lee HS: Urinary tract dysfunction after radical hysterectomy for cervical cancer. GynecolOncol 2002; 85: 292-297

73. Glahn BE: The neurogenic factor in vesical dysfunction following radical hysterectomy for carcinoma of the cervix. Scand J UrolNephrol 1970; 4:107-116.

74. Green TH Jr, Meigs JV, Ulfelder H, Curtin RR: Urologic complications of radical Wertheim hysterectomy: Incidence, etiology, management, and prevention. ObstetGynecol 1962; 20:293-312.

75. O'Callaghan PL, Da D: Urinary dysfunction following radical hysterectomy; Is there spontaneous improvement with time? NeurourolUrodyn 1993;12(Suppl):429

76. Zullo MA, Manci N, Angioli R, Muzii L, Panici PB: Vesical dysfunctions after radical hysterectomy for cervical cancer: A critical review. Crit Rev OncolHematol 2003; 48:287-293.

77. Landoni F, Maneo A, Cormio G, et al: Class II versus class III radical hysterectomy in stage IB-IIA cervical cancer: A prospective randomized study. GynecolOncol 2001; 80:3-12.
78. Yabuki Y, Asamoto A, Hoshiba T, Nishimoto H, Nishikawa Y, Nakajima T: Radical hysterectomy: An anatomic evaluation of parametrial dissection. GynecolOncol 2000; 77:155-163.
79. Sakuragi N, Todo Y, Kudo M, Yamamoto R, Sato T: A systematic nerve-sparing radical hysterectomy technique in invasive cervical cancer for preserving postsurgical bladder function. Int J Gynecol Cancer 2005; 15:389-397.
80. Raspagliesi F, Ditto A, Fontanelli R, et al: Type II versus Type III nerve-sparing radical hysterectomy: Comparison of lower urinary tract dysfunctions. GynecolOncol 2006; 102:256-262.
81. Todo Y, Kuwabara M, Watari H, et al: Urodynamic study on postsurgical bladder function in cervical cancer treated with systematic nerve-sparing radical hysterectomy. Int J Gynecol Cancer 2006; 16: 369-375.
82. EEspinoStrebelEE , Luna JT, Domingo EJ. A comparison of the feasibility and safety of nervesparing radical hysterectomy with the conventional radical hysterectomy. Int J Gynecol Cancer. 2010 Oct;20(7):127483.
83. Ditto A ,Martinelli F, Mattana F, Reato C, Solima E, Carcangiu M, Haeusler E, Mariani L, Raspagliesi F. Class III nervesparing radical hysterectomy versus

standard class III radical hysterectomy: an observational study. Ann SurgOncol. 2011 Nov;18 (12):3469 -78.

84. Y. Long, D.-S. Yao, X.-W. Pan, and T.-Y. Ou, "Clinical efficacy and safety of nerve-sparing radical hysterectomy for cervical cancer: a systematic review and meta-analysis," PLoS ONE, vol. 9, no. 4, Article IDe94116, 2014.
85. N. Y. Park, G. O. Chong, D. G. Hong, Y. L. Cho, I. S. Park, and Y. S. Lee, "Oncologic results and surgical morbidity of laparoscopic nerve-sparing radical hysterectomy in the treatment of figo stage IB cervical cancer: long-term follow-up," International Journal of Gynecological Cancer, vol. 21, no. 2, pp. 355–362, 2011.
86. T. Zhu, A. J. Yu, H. F. Shou et al., "Feasibility of unilateral or bilateral nerve-sparing radical hysterectomy in patients with cervical cancer and evaluation of the post-surgery recovery of the bladder and rectal function," Chinese Journal of Oncology, vol. 33, no. 1, pp. 53–57, 2011.
87. M. Eicke, R. Hohenfellner, and J. Leissner, "Nerve injuries in urologic surgery," in Advanced Urologic Surgery, vol. 75, pp. 488–493, Blackwell, 2005.
88. D. W. Zochodne, "Nerve and ganglion blood flow in diabetes: an appraisal," International Review of Neurobiology, vol. 50, pp. 161–202, 2002.
89. G. Ralph, K. Tamussino, and W. Lichtenegger, "Urodynamics following radical abdominal hysterectomy for cervical cancer," Archives of Gynecology and Obstetrics, vol. 243, no. 4, pp. 215– 220, 1988.

90. Lin YS: Preliminary results of laparoscopic modified radical hysterectomy in early invasive cervical cancer. J Am AssocGynecolLaparosc 2003; 10:80-84
91. Nam JH, Park JY, Kim DY, Kim JH, Kim YM, Kim YT. Laparoscopic versus open radical hysterectomy in early-stage cervical cancer: long-term survival outcomes in a matched cohort study. Ann Oncol 2012; 23:903-11.
92. Park NY, Chong GO, Hong DG, Cho YL, Park IS, Lee YS. Oncologic results and surgical morbidity of laparoscopic nerve-sparing radical hysterectomy in the treatment of FIGO stage IB cervical cancer: long-term follow-up. Int J Gynecol Cancer 2011;21:355-62.
93. Hiroyuki Kanao, Kazuko Fujiwara, Keiko Ebisawa, Tomonori Hada, Yoshiaki Ota, Masaaki Andou Department of Obstetrics and Gynecology, Kurashiki Medical Center, Kurashiki, Japan. Various types of total laparoscopic nerve-sparing radical hysterectomies and their effects on bladder function. J GynecolOncol Vol. 25, No. 3:198-205
94. Andersson KE. Bladder activation: afferent mechanisms. Urology 2002; 59 (5 Suppl 1):43-50.
95. Ramirez PT, Adams S, Boggess JF, Burke WM, Frumovitz MM, Gardner GJ, et al. Robotic-assisted surgery in gynecologic oncology: a Society of Gynecologic Oncology consensus statement. Developed by the Society of Gynecologic Oncology's Clinical Practice Robotics Task Force. GynecolOncol 2012; 124 : 180-4.

96. Sarlos D, Kots L, Stevanovic N, Schaer G. Robotic hysterectomy versus conventional laparoscopic hysterectomy: outcome and cost analyses of a matched case-control study. Eur J ObstetGynecolReprodBiol 2010; 150:92-6.

97. Hoekstra AV, Morgan JM, Lurain JR, Buttin BM, Singh DK, Schink JC, et al. Robotic surgery in gynecologic oncology: impact on fellowship training. GynecolOncol 2009;114:168-72.

98. O'Neill M, Moran PS, Teljeur C, O'Sullivan OE, O'Reilly BA, Hewitt M, et al. Robot-assisted hysterectomy compared to open and laparoscopic approaches: systematic review and meta-analysis. Arch GynecolObstet 2013; 287:907-18.

99. Drach et al., 1979b. Drach GW, Layton TN, Binard WJ: Male peak urinary flow rate: Relationships to volume voided and age. J Urol 1979; 122:210-214.

100. Jorgensen et al., 1986. Jorgensen JB, Jensen KM, Bille-Brahe NE, Morgensen P: Uroflowmetry in asymptomatic elderly males. Br J Urol 1986; 58:390-395.

101. Bottaccini and Gleason, 1980. Bottaccini MR, Gleason DM: Urodynamic norms in women. I. Normals versus stress incontinents. J Urol 1980; 124:659-662.

102. Schafer et al., 2002. Schafer W, Abrams P, Liao L, et al: Good urodynamic practices: Uroflowmetry, filling cystometry, and pressure-flow studies. NeurourolUrodyn 2002; 21:261-274.

103. Chancellor et al., 1991. Chancellor MB, Blaivas JG, Kaplan SA, Axelrod S: Bladder outlet obstruction versus impaired detrusor contractility: The role of outflow. J Urol 1991; 145:810-812.

104. HakenbergOwmRyall RL, Langlois SL, Marshall VR. The estimation of bladder volume by sonocystography. J Urol 1983; 130:249-51.

105. Low JA, Mauger GM, Carmichael JA. The effect of Wertheim hysterectomy upon bladder and urethral function. Am J ObstetGynecol 1981; 139:826-34.

106. Scotti RJ, Bergman A, Bhatia NN, Ostergard DR. Urodynamic changes in urethrovesical function after radical hysterectomy. ObstetGynecol 1986;68:111-20.

107. Ralph G, Winter R, Michelitsch L, Tamussino K. Radicality of parametrial resection and dysfunction of the lower urinary tract after radical hysterectomy. Eur J GynaecolOncol 1991; 12:27-30.

108. Sekido N, Kawai K, Akaza H. Lower urinary tract dysfunction as persistent complication of radical hysterectomy. Int J Urol 1997;4: 259-64.

109. Lin HH, Sheu BC, Lo MC, Huang SC. Abnormal urodynamic findings after radical hysterectomy or pelvic irradiation for cervical cancer. Int J GynaecolObstet 1998; 63:169-74.

110. Chuang FC, Kuo HC. Management of lower urinary tract dysfunction after radical hysterectomy with or without radiotherapy for uterine cervical cancer. J Formos Med Assoc 2009; 108:619-26.

111. Jin Kyu Oh, Min SooChoo ,Joongyub Lee , Noh-Hyun Park , Seung-June Oh. Short-term Effect of Radical Hysterectomy with or without Adjuvant Radiation Therapy on Urodynamic Parameters in Patients with Uterine Cervical Cancer. IntNeurourol J 2012;16:91-95

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Ref.No.12117/ME-1/Ethics/2012 Dt:03.01.2013.
CERTIFICATE OF APPROVAL

The Institutional Ethical Committee of Govt. Kilpauk Medical College, Chennai reviewed and discussed the application for approval "A Study on a prospective study on urinary bladder morbidity following pelvic surgery" for dissertation purpose submitted by Dr.P.Senthilkumar, MCh (Surgical Oncology), PG Student, Govt. Kilpauk Medical College, Chennai.

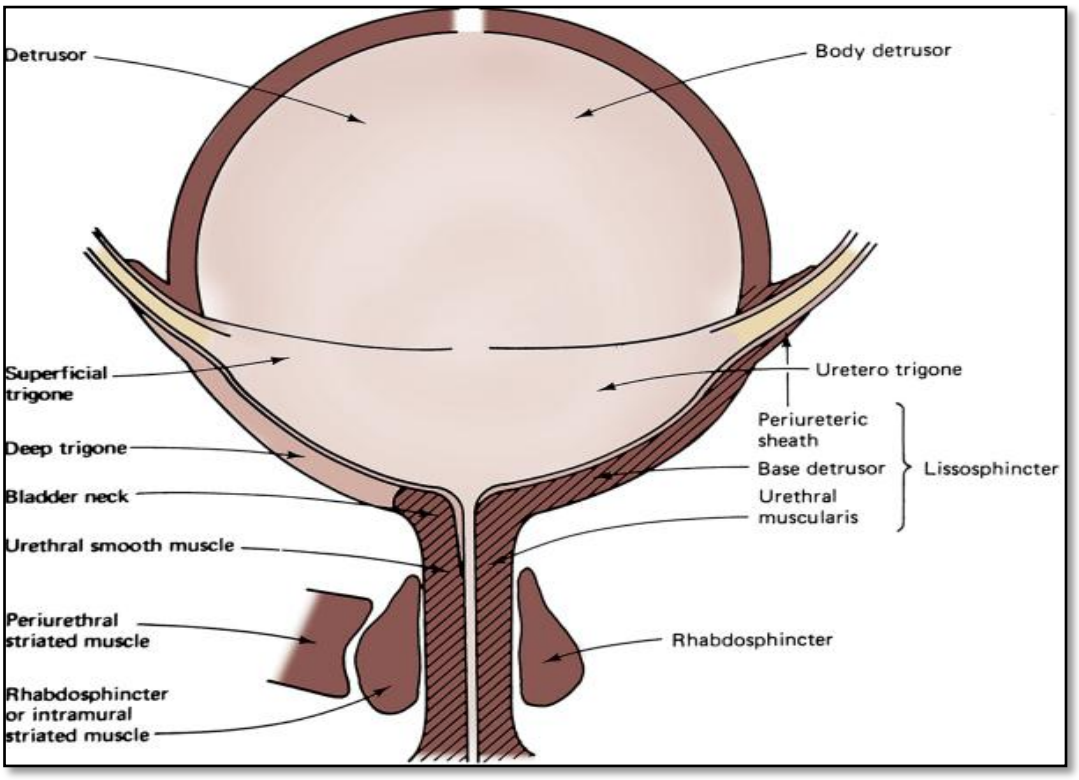
The Proposal is APPROVED.

The Institutional Ethical Committee expects to be informed about the progress of the study any Adverse Drug Reaction Occurring in the Course of the study any change in the protocol and patient information /informed consent and asks to be provided a copy of the final report.

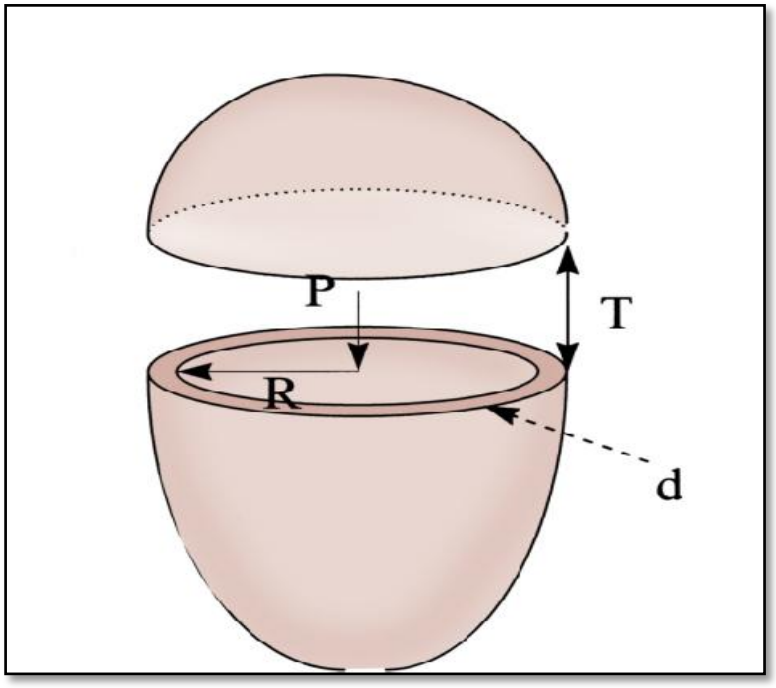
CHAIRMAN, 21/1/13.
Ethical Committee
Govt.Kilpauk Medical College,Chennai

ANATOMY OF LOWER URINARY TRACT

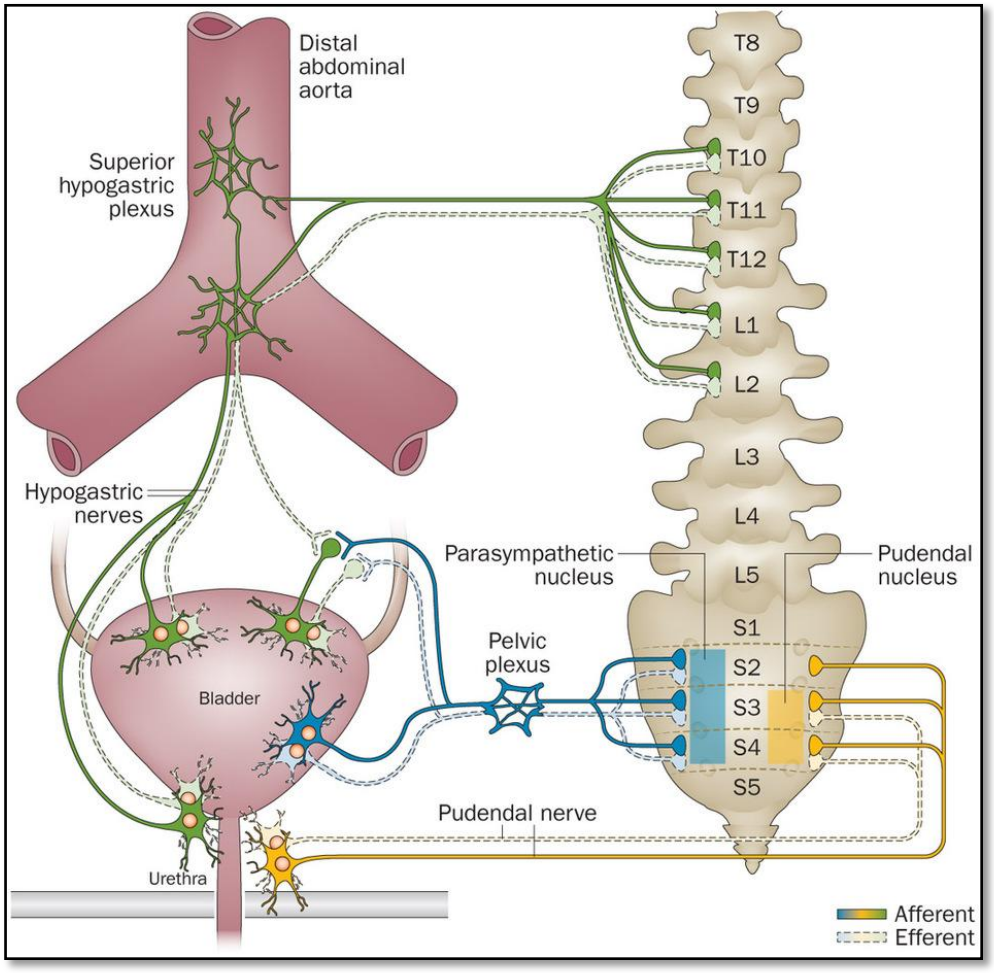
Left– Gosling & Dixon; Right- Elbadawi & Coworkers



Laplace's Law T = P ×R ÷ (2×d)



INNERVATION OF LOWER URINARY TRACT



- Sympathetic nerve fibers originate from the para-aortic sympathetic chain of the thoracic and lumbar nerve roots T10–L2. Postganglionic nerve fibers run to the superior hypogastric plexus, connect it with the pelvic plexuses, and innervate the bladder, bladder neck and urethra.

- The parasympathetic splanchnic nerves originate from the sacral nerve roots S2–S4 and run through the pelvic plexuses to the bladder.

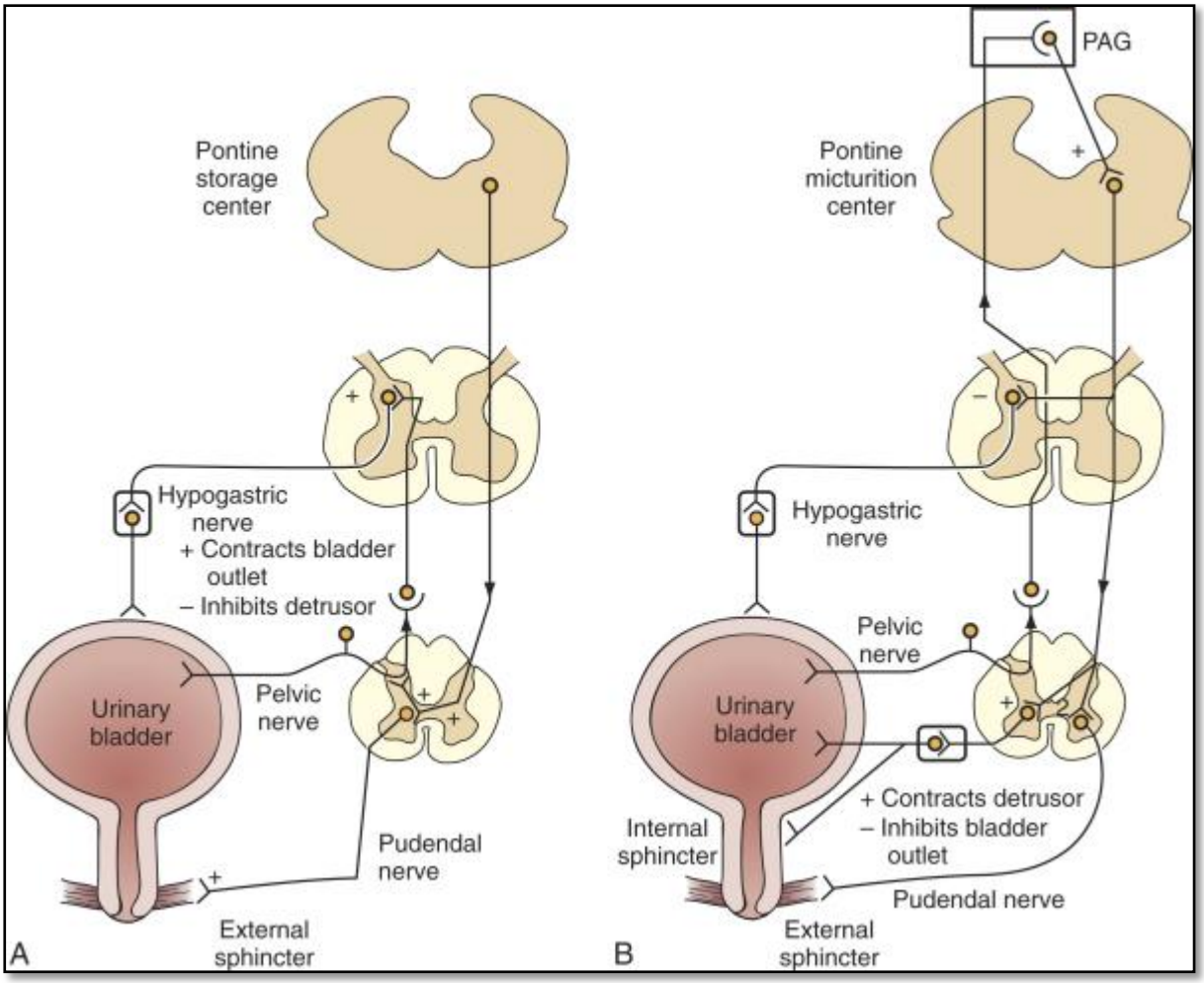
- In the pelvic plexuses, parasympathetic nerves synapse with sympathetic fibres.

- The pudendal nerve innervates the external urethral sphincter with sensory and motor fibers.

Mechanism of storage and voiding reflexes

A - Storage reflexes

During the storage of urine, distension of the bladder produces low-level bladder afferent firing. Afferent firing in turn stimulates the sympathetic outflow to the bladder outlet (base and urethra) and pudendal outflow to the external urethral sphincter. These responses occur by spinal reflex pathways and represent "guarding reflexes," which promote continence. Sympathetic firing also inhibits detrusor muscle and transmission in bladder ganglia

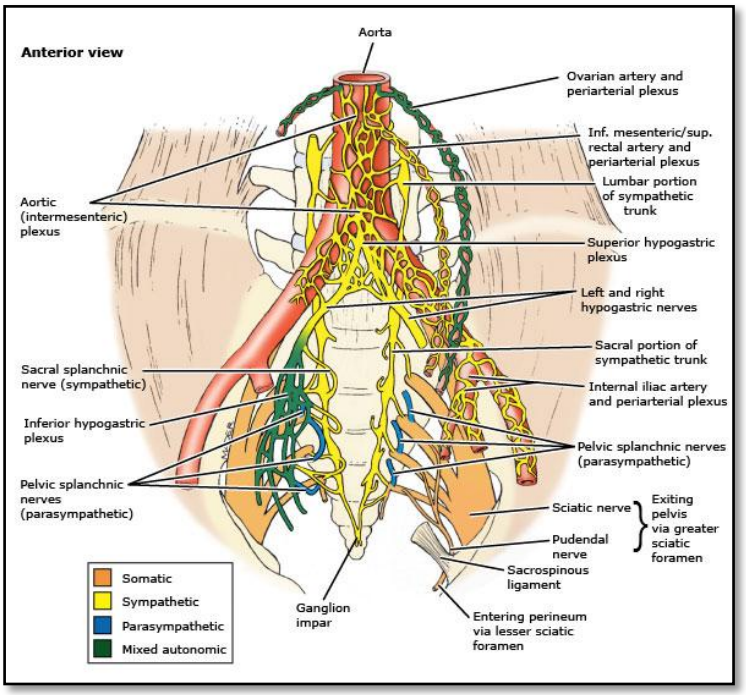


B -Voiding reflexes

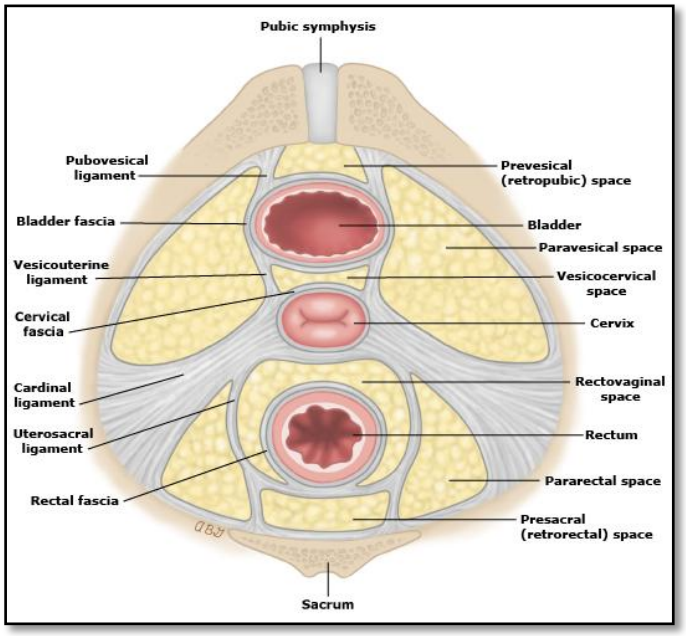
At the initiation of micturition, intense vesical afferent activity activates the brainstem micturition centre, which inhibits the spinal guarding reflexes (sympathetic and pudendal outflow to the urethra). The pontine micturition centre also stimulates the parasympathetic outflow to the bladder and internal sphincter smooth muscle. Maintenance of the voiding reflex is through ascending afferent input from the spinal cord, which may pass through the periaqueductal gray matter (PAG) before reaching the pontine micturition centre.

Autonomic Nerve Plexus of Pelvis

The superior hypogastric plexus is a continuation of the aortic (inter-mesenteric) plexus. It divides into left and right hypogastric nerves as it enters the pelvis. The hypogastric and pelvic splanchnic nerves merge to form the inferior hypogastric plexuses, which thus consist of both sympathetic and parasympathetic fibres.



Avascular Spaces of Pelvis



The bladder, cervix, and rectum are surrounded by a connective tissue covering. Between the firm connective tissue bundles is loose connective tissue (Para spaces).

CLASSIFICATION OF RADICAL HYSTERECTOMY ACCORDING TO PIVER, RUTLEDGE AND SMITH

Parametrial dissection in Type I, II, & III

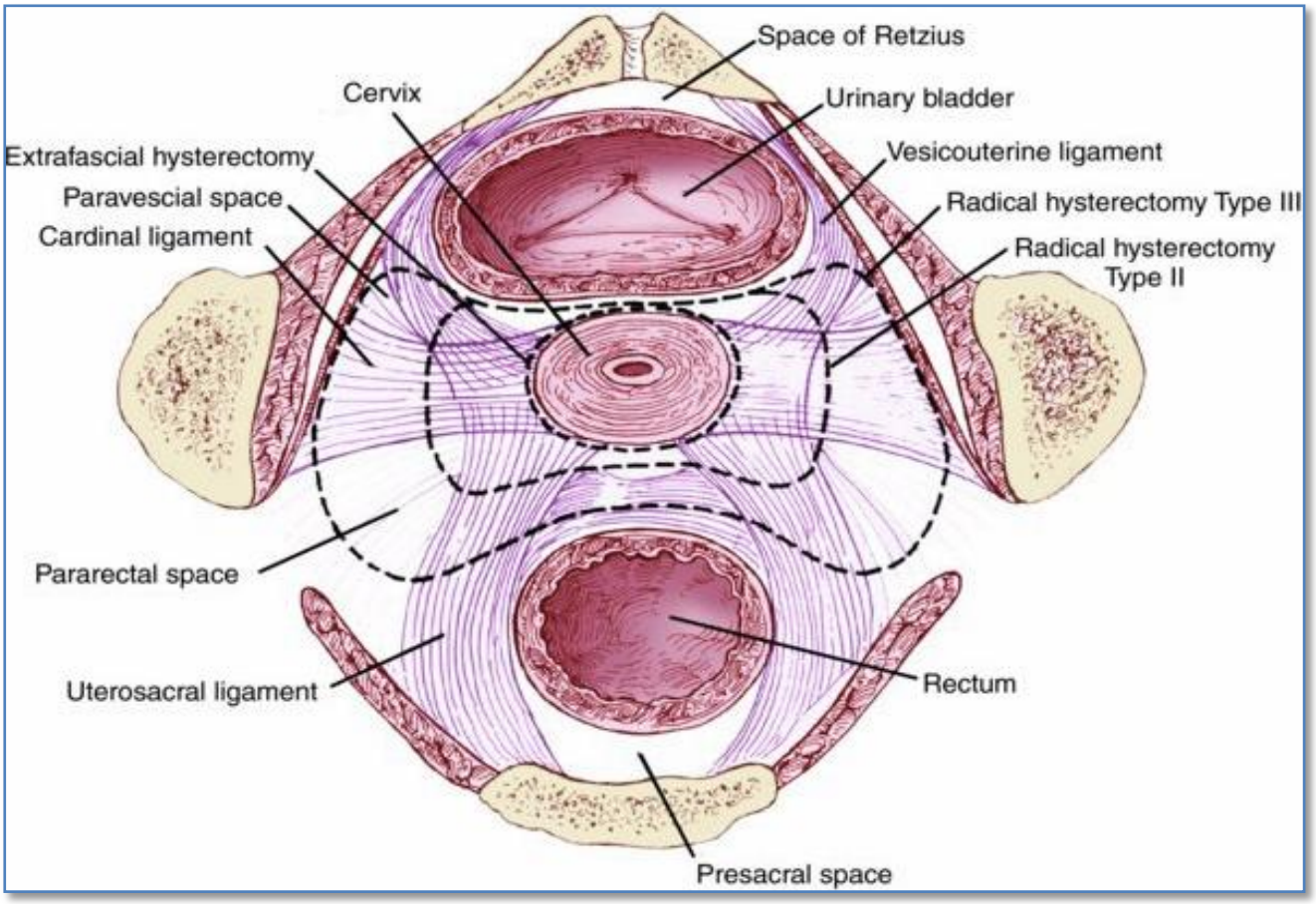
Type I

Uterosacral ligament and cardinal ligament are not removed

Type II

Uterosacral ligaments resected midway between the uterus and their sacral attachments

Medial half of the cardinal ligaments removed

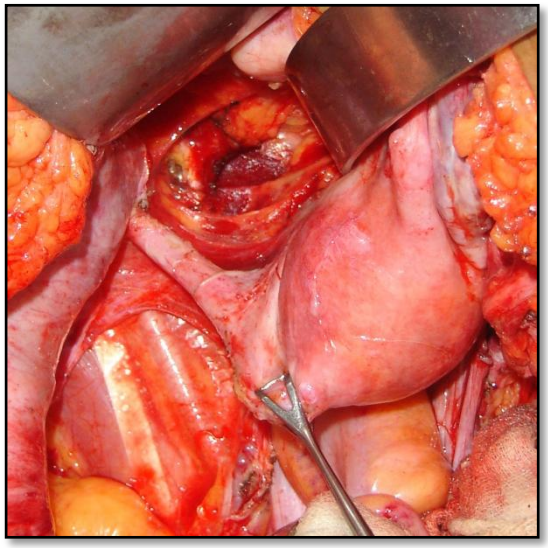


TYPE III

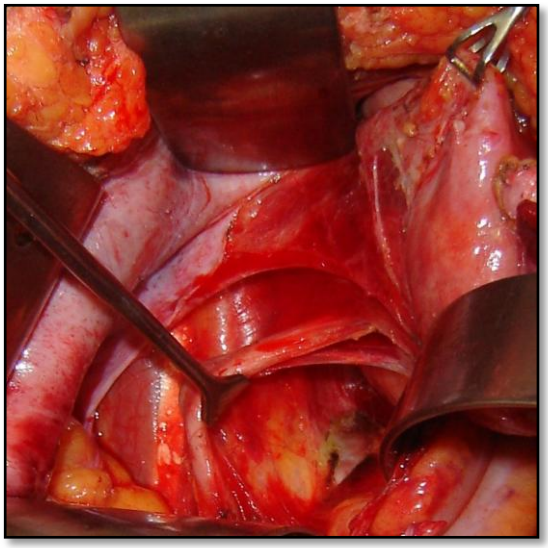
Uterosacral ligaments resected at their sacral attachments

Cardinal ligaments resected at the pelvic wall

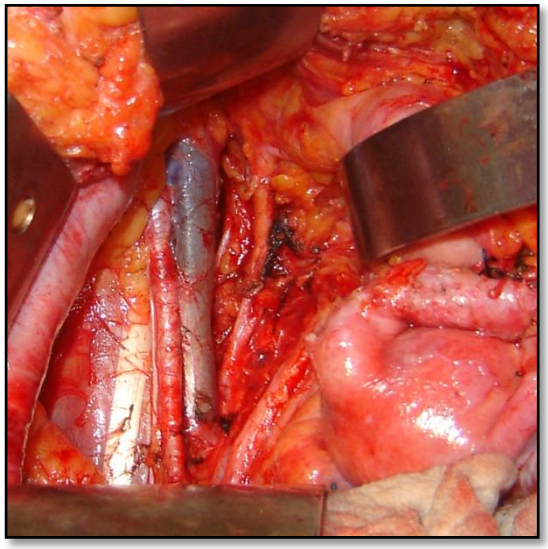
OPERATIVE PICTURES – TYPE III RADICAL HYSTERCETOMY



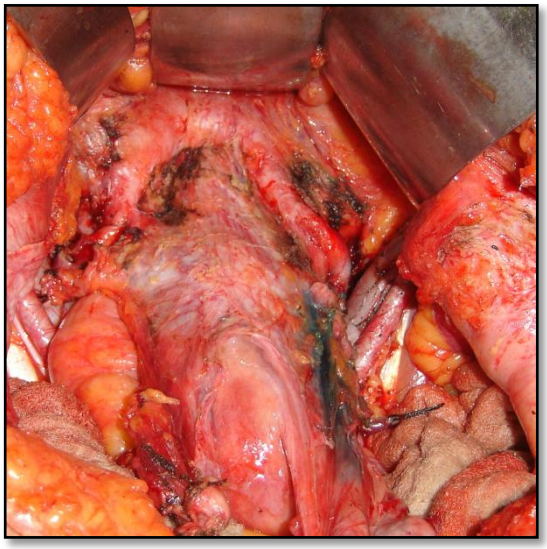
Paravesical Fossa



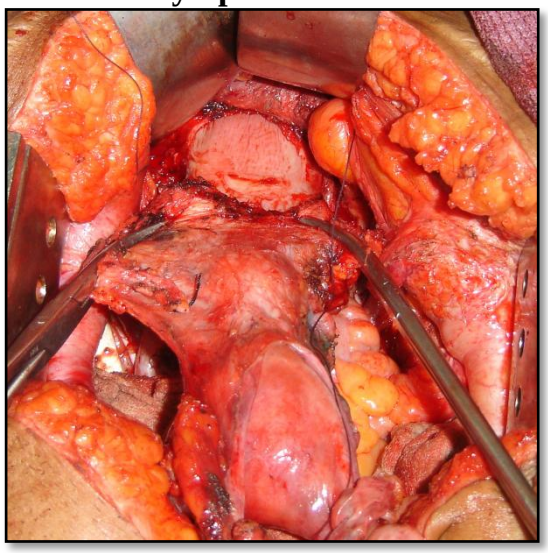
Pararectal Fossa



Pelvic lymph node dissection



Ureterovesical junction

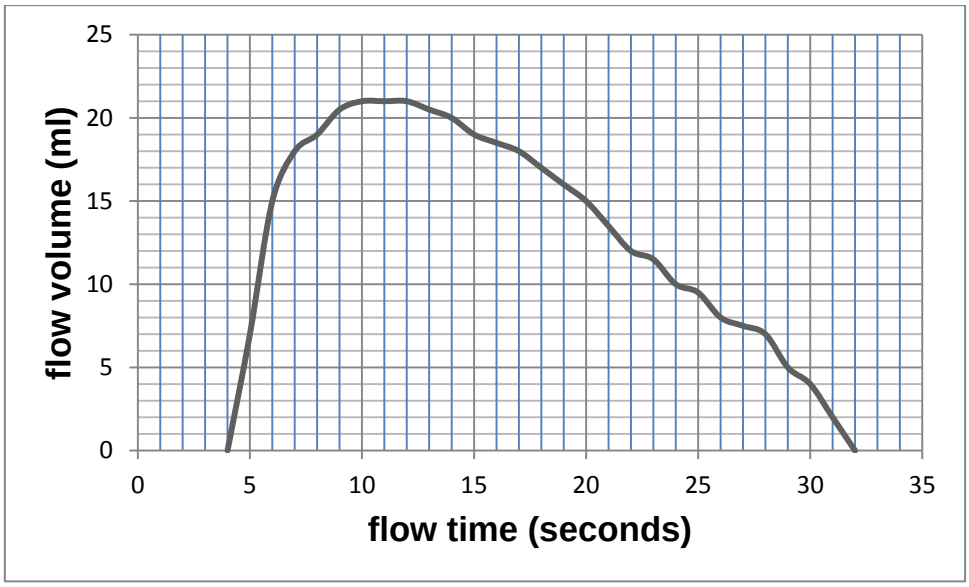


Parametrium

Specimen

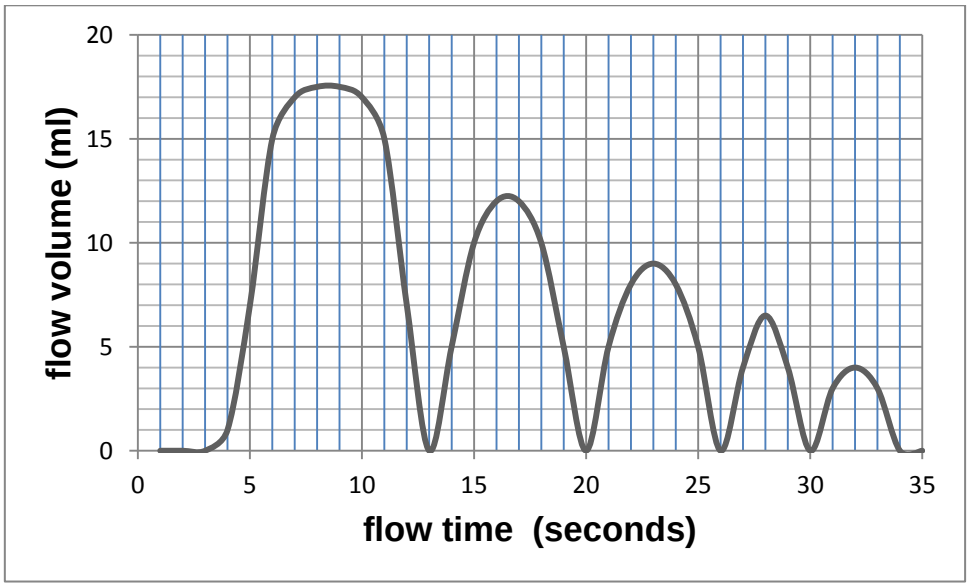
Normal uroflow curve

Continuous, bell-shaped, smooth curve with a rapidly increasing flow rate



Abnormal uroflow curve

An intermittent flow pattern is one that has several episodes of flow increasing or decreasing (or ceasing completely) and is commonly secondary to abdominal straining or external sphincter spasm



MASTER CHART

ABBREVATIONS	
CD	Cancer Disease Number
D	Diagnosis C- Cervix E-Endometrium
S	Stage
Wt	Weight in Kilogram
Ht	Height in meter
Pre OP	Preoperative Therapy 1=Nil- Upfront surgery 2=PreOp Chemotherapy 3= PreOp Radiotherapy 4= PreOp Chemoradiation 5= Definitive Chemoradiation
Adj	Adjuvant Therapy 1= External Beam Radio therapy 2= Vaginal Brachytherapy 3= External Radiotherapy + Vaginal Brachytherapy
U	Intra-operative ureteric injury
UB	Intra-operative urinary bladder injury
SSI	Surgical Site Infection
M	Mortality
F	Post-operative urinary fistula
P	Parametrium 0 – negative , 1 - positive
V	Vaginal margin 0 – negative , 1 - positive
N	Pelvic node 0 – negative , 1 - positive
r	Recurrence within 6^{th} months
B	Preoperative Baseline Value
m	Post-operative month
Uroflow curve 0- normal , 1- abnormal	
Voiding Symptoms 0- absent , 1- present	

Master Chart 1

S. No	CD No	Name	Age	D	S	Pre Op	Adj	Wt Kg	Ht m	BMI	U	UB	M	SSI	F	P	V	N	r
1	1085/12	Malliga	60	C	IIB	2	0	45	1.62	17.1	0	0	0	0	0	0	0	0	0
2	1018/12	Kasthuri	53	C	IIB	2	2	45	1.65	16.5	0	0	0	0	0	0	0	0	0
3	109/13	Murugammal	60	C	IB 1	1	1	48	1.53	20.5	0	0	0	0	0	0	0	0	0
4	873/12	Valliammal	36	C	IIB	4	0	47	1.52	20.3	0	0	0	1	0	0	0	0	0
5	315/13	Chinnaponnu	55	C	IIB	4	0	50	1.53	21.4	0	0	0	0	0	0	0	0	0
6	1378/12	Muthmari	36	C	IIB	2	2	52	1.54	21.9	0	0	0	0	0	0	0	0	0
7	25/13	Sumathy	40	C	IB 2	2	2	53	1.57	21.5	0	0	0	0	0	0	0	0	0
8	1350/12	Alamelu	36	C	IIB	2	2	56	1.64	20.8	0	0	0	0	0	0	0	0	0
9	409/13	Shanthi	52	E	II	1	3	57	1.53	24.3	0	0	0	0	0	0	0	0	0
10	493/13	Rani	49	C	IIA 1	3	0	54	1.63	20.3	0	0	0	0	0	0	0	0	0
11	195/13	Ramisha	55	C	IIB	3	0	45	1.57	18.3	0	0	0	0	0	0	0	0	0
12	1394/13	Mala	45	C	IB 2	2	2	54	1.58	21.6	0	0	0	0	0	0	0	0	0
13	637/13	Shanthi	34	C	IB 1	1	0	57	1.56	23.4	0	0	0	0	0	0	0	0	0
14	361/13	Shantha	38	C	IIB	2	2	51	1.54	21.5	0	0	0	0	0	0	0	0	0
15	645/13	Kilayammal	60	C	IIA 1	1	1	45	1.46	21.1	0	0	0	0	0	0	0	0	0
16	756/13	Loganayaki	57	C	IB 1	1	0	47	1.72	15.9	0	0	0	0	0	0	0	0	0
17	955/12	Vijaya	42	C	IIB	4	0	48	1.73	16.0	0	0	0	0	0	0	0	0	0
18	248/13	Vedavalli	50	C	IIB	4	0	45	1.71	15.4	0	0	0	1	0	0	0	0	0
19	984/13	Nagamal	50	C	IB 1	1	0	46	1.47	21.3	0	0	0	0	0	0	0	0	0
20	601/13	Rajakodi	55	C	IIA 2	2	0	45	1.48	20.5	0	0	0	1	0	0	0	0	0
21	739/12	Shakunthala	55	C	IB 2	4	0	49	1.52	21.2	0	0	0	0	0	0	0	0	0
22	850/13	Kottesawari	43	C	IIB	4	0	54	1.67	19.4	0	0	0	0	0	0	0	0	0
23	1176/13	Palaniammal	48	C	IIA 2	5	0	54	1.7	18.7	0	0	0	0	0	0	0	0	0
24	996/13	Lakshmi	38	C	IIB	4	0	54	1.71	18.5	0	0	0	0	0	0	0	0	0
25	1013/13	Rani	48	C	IIB	4	0	55	1.72	18.6	0	0	0	0	0	0	0	0	0
26	1409/13	Kavitha	37	C	IIB	4	0	49	1.53	20.9	0	0	0	1	0	0	0	0	0

Master Chart 1 Contd.

S. No	Voided Volume (mL)				Maximum Flow Rate (mL/s)				Average Flow Rate (mL/s)				Post-Void Residual Urine (mL)				Uroflow curve			
	B	1m	3m	6m	B	1m	3m	6m	B	1m	3m	6m	B	1m	3m	6m	B	1m	3m	6m
1	310	240	320	300	18.8	18.9	18.7	18.7	10.7	8.5	9	9	30	60	45	0	0	1	1	0
2	290	300	250	300	18.8	19	18.7	18.6	10.2	10.4	10.3	10.3	0	0	30	30	0	0	0	0
3	250	240	290	240	24.2	23.9	23.8	24.1	10.7	10.8	10.9	10.8	30	30	35	40	0	0	0	0
4	240	280	250	220	18.8	18.9	19.2	18.7	10.1	10.4	10.3	10.3	30	0	45	30	0	0	0	0
5	310	320	280	310	22.7	19.5	18.8	22.8	10.6	8.9	9.6	10.8	35	0	30	0	0	0	0	0
6	270	270	300	260	22.6	22.7	20.5	23.4	10.5	8.7	9.4	10.6	35	0	35	35	0	0	0	0
7	250	200	210	250	22.7	19.3	18.8	22.6	9.6	9.7	9.7	9.8	0	60	0	0	0	1	0	0
8	270	280	280	250	22.2	22.3	22.1	22.1	9.1	9.2	9.5	9.6	30	75	60	30	0	1	0	0
9	310	320	220	230	21.2	20.5	21	20.8	10.3	10.1	9.9	10	30	60	30	60	0	0	0	0
10	290	310	210	300	22.2	22	22.3	22.1	10.5	10.8	10.7	10.6	0	0	30	45	0	0	0	0
11	310	230	220	320	23	19.1	20.6	22.9	10	9.7	10.1	9.8	30	0	70	40	0	0	1	1
12	280	290	310	300	20.2	20.4	20.1	18.8	10	10.1	10.2	9.4	30	30	45	0	0	0	0	0
13	280	290	280	290	22.7	23.4	22.6	18.8	10.4	9	9.3	9.5	30	0	0	40	0	0	0	1
14	300	220	220	310	24.8	20.2	24.9	24.6	10.3	8.5	9.5	10.4	30	0	0	60	0	0	0	0
15	260	270	310	270	23.9	22.4	24	23.7	11.1	9.5	10.5	10	35	45	0	0	0	1	1	0
16	240	260	250	230	22.2	22.3	23.5	22.3	11	9.1	9.9	10	35	0	60	0	0	0	0	0
17	280	290	300	290	20.8	22.5	20.5	20.1	10.1	10.2	10.2	10.3	35	0	35	35	0	1	1	0
18	300	220	230	300	24.8	20.2	21.9	23.2	10	10.1	10.2	9.4	0	100	30	30	0	1	0	0
19	250	260	270	260	24.8	24.9	24.7	24.9	10.3	10.4	9.7	9.8	0	60	0	0	0	1	0	0
20	280	220	230	280	21.5	21.7	21.7	21.3	9.8	9.9	10.2	10.1	0	100	30	30	0	1	0	0
21	290	300	260	280	23.9	23.8	21.3	21.4	10.3	8.5	10.3	10.4	0	30	0	0	0	0	0	0
22	300	310	310	310	21.5	20.3	20.5	22	11.1	9.3	9.9	10	45	60	35	30	0	1	0	0
23	260	270	270	280	20.2	20.4	20.4	18.8	10.6	10.7	9.8	10.7	0	30	0	0	0	0	0	0
24	270	280	260	260	25	22.6	22.6	24.9	11.3	9.5	10	9.8	30	45	30	30	0	0	0	0
25	300	310	320	320	21.9	21.7	21.5	21.7	10.6	8.5	9	9.2	0	30	0	0	0	0	0	0
26	280	270	290	270	18.6	18.9	18.5	19.2	10.3	10.4	10.4	10.4	30	0	0	35	0	0	0	1

Master Chart 1 Contd.

S. No	Straining				Incontinence				Urgency				Dysuria				Overall Symptoms				Needed Catheterization			
	B	1m	3m	6m	B	1m	3m	6m	B	1m	3m	6m	B	1m	3m	6m	B	1m	3m	6m	B	1m	3m	6m
1	0	1	0	0	0	1	0	0	0	1	0	0	0	0	0	0	0	1	0	0	0	0	0	0
2	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
3	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
4	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
5	0	1	0	0	0	1	0	0	0	1	0	0	0	1	0	0	0	1	0	0	0	0	0	0
6	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
7	0	1	0	0	0	1	0	0	0	1	0	0	0	1	0	0	0	1	0	0	0	0	0	0
8	0	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	1	1	1	0	1	0	0
9	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
10	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
11	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0
12	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
13	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0
14	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
15	0	1	1	0	0	1	1	0	0	1	1	0	0	0	0	0	0	1	1	0	0	0	0	0
16	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
17	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
18	0	1	1	0	0	1	1	0	0	0	0	0	0	1	1	0	0	1	1	0	0	1	0	0
19	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
20	0	1	1	0	0	1	1	0	0	0	0	0	0	1	0	0	0	1	1	0	0	1	0	0
21	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
22	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0
23	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
24	0	1	1	1	0	1	1	1	0	1	1	0	0	0	0	0	0	1	1	1	0	0	0	0
25	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
26	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0

.

Master Chart 2

S. No	CD No	Name	Age	D	S	Pre Op	Adj	Wt Kg	Ht m	BMI	U	UB	M	SSI	F	P	V	N	r
27	956/13	Backiyalakshmi	45	C	IIA 2	4	0	53	1.53	22.6	0	0	0	0	0	0	0	0	0
28	1524/13	Bakiyam	60	C	IB 1	1	0	52	1.54	21.9	0	0	0	0	0	0	0	0	0
29	1024/13	Kuppu	55	C	IIB	2	0	51	1.55	21.2	0	0	0	0	0	0	0	0	0
30	1317/13	Munniammal	48	C	IIB	4	0	55	1.56	22.6	0	0	0	0	0	0	0	0	0
31	115/14	Rajeswari51	51	C	IB 1	1	0	47	1.57	19.1	0	0	0	1	0	0	0	0	0
32	1323/13	Jothi	45	C	IIB	2	2	46	1.58	18.4	0	0	0	0	0	0	0	0	0
33	186/14	Saraswathy	40	C	IB 1	1	0	48	1.61	18.5	0	0	0	0	0	0	0	0	0
34	298/14	Jileeka Bee	45	E	II	1	0	49	1.6	19.1	0	0	0	0	0	0	0	0	0
35	18/14	Muniammal	50	C	IIB	4	0	54	1.47	25.0	0	0	0	1	0	0	0	0	0
36	1524/13	Chockamal50	50	C	IB 2	2	0	50	1.52	21.6	0	0	0	0	0	0	0	0	0
37	356/14	Saroja	55	C	IB 1	1	0	49	1.55	20.4	0	0	0	0	0	0	0	0	0
38	60/14	Sivamma	45	C	IB 2	2	0	51	1.56	21.0	0	0	0	0	0	0	0	0	0
39	215/14	Tamilarassa	47	C	IIB	5	0	48	1.62	18.3	0	0	0	0	0	0	0	0	0
40	674/14	Lingammal	64	E	II	1	2	53	1.65	19.5	0	0	0	0	0	0	0	0	1
41	700/14	Kamatchi	60	E	II	1	0	51	1.56	21.0	0	0	0	0	0	0	0	0	0
42	121/14	Kannamal	68	C	IIB	4	0	55	1.48	25.1	0	0	0	0	0	0	0	0	0
43	378/14	Muniammal	56	C	IIB	2	0	65	1.53	27.8	0	0	0	0	0	0	0	0	0
44	780/14	Ruksana	32	C	IIA 2	3	0	50	1.57	20.3	0	0	0	0	0	0	0	0	0
45	781/14	Rasiyamani	60	E	II	1	3	54	1.53	23.1	0	0	0	1	0	0	0	0	0
46	412/14	Seetha	68	C	IIB	2	2	52	1.64	19.3	0	0	0	0	0	0	0	0	0
47	690/13	Deivani	60	C	IIB	5	0	48	1.6	18.8	0	0	0	1	0	0	0	0	0
48	654/14	Kodiyarasi	37	C	IIB	4	0	48	1.55	20.0	0	0	0	0	0	0	0	0	0
49	983/14	Periyammal	40	C	IB 1	1	0	67	1.63	25.2	0	0	0	0	0	0	0	0	0
50	1110/14	Lakshmi	69	C	IIB	3	0	49	1.54	20.7	0	0	0	0	0	0	0	0	0
51	988/14	Rani	55	C	IB 1	1	0	58	1.52	25.1	0	0	0	0	0	0	0	0	0

Master Chart 2 Contd.

S. No	Voided Volume (mL)				Maximum Flow Rate (mL/s)				Average Flow Rate (mL/s)				Post-Void Residual Urine (mL)				Uroflow curve			
	B	1m	3m	6m	B	1m	3m	6m	B	1m	3m	6m	B	1m	3m	6m	B	1m	3m	6m
27	260	210	220	240	19.8	17.2	19.9	20.8	10.9	9	9.8	10	35	0	0	0	0	0	0	0
28	290	230	300	280	23	23.8	23.4	23.4	10	10.1	10.2	9.9	0	55	0	0	0	0	0	0
29	250	210	220	240	25	25.4	25.1	24.8	10.5	10.6	10.7	10.4	35	0	0	30	0	0	1	0
30	309	264	310	311	19.5	17	18.6	16.5	10.3	10.4	10.3	9.8	0	55	0	35	0	1	0	0
31	260	270	250	250	21.9	22.1	19.7	21.5	10.2	10.3	10.1	10.4	45	75	30	35	0	1	0	0
32	250	240	260	270	20	18.2	18.7	17.5	10.8	10.9	9.8	10.9	0	85	0	0	0	1	0	0
33	280	290	300	290	18.6	18.9	17.3	18.8	9.9	10	10.1	9.8	30	30	45	30	0	0	0	0
34	310	300	320	290	21.8	20.8	21.8	21.6	10.5	9.9	10	10.1	30	45	30	0	0	0	0	0
35	300	260	290	310	21.9	18.7	22.1	21.7	10	10.1	10.1	10.1	30	30	55	30	0	0	0	0
36	290	220	230	280	19.5	17	16.5	18.6	10.9	9.4	9.5	11	30	30	30	30	0	0	0	0
37	280	250	270	290	21.9	22.1	22	22.1	10.2	10.5	10.4	10.3	0	100	40	55	0	0	0	1
38	250	270	240	260	22	21.9	22.1	22.3	9.9	10	10.2	10.1	30	0	55	30	0	0	0	0
39	260	270	280	270	22.3	18.2	22.1	22	10	10.2	10.2	10.1	35	100	60	60	0	1	0	0
40	290	310	300	270	20.5	21.4	20.6	20.6	9.9	10	10.1	10.1	45	30	45	30	0	0	0	0
41	310	270	300	290	19.8	20.2	19.9	20.4	10	10.1	10.1	10.2	0	45	0	0	0	0	0	0
42	290	300	300	280	20.6	20.5	20.8	20.5	10.3	10.4	10.6	10.4	35	45	35	35	0	0	0	0
43	260	270	250	250	25.2	18.9	25.6	25.7	10.3	10.4	10.4	10.5	0	50	0	0	0	0	0	0
44	290	230	300	290	19.4	20.8	19.6	20	10.2	10.3	10.3	10.4	0	30	0	0	0	0	0	0
45	280	250	290	300	22.1	21.4	21.6	22	10	9.9	10	10	30	0	0	0	0	0	0	0
46	290	300	270	270	22.4	18.7	22.3	22.5	11.3	9.6	9.4	9.9	30	0	35	30	0	0	0	0
47	310	250	320	300	18.3	19.2	18.3	18.2	10	10.1	10.1	9.8	30	90	55	30	0	1	1	0
48	310	240	280	320	23.6	17.9	18.5	20	10.9	9.4	10.1	11	0	55	30	30	0	1	0	0
49	280	290	300	270	22.4	18.7	22.5	22.6	10	10.1	10.3	10.2	35	0	35	35	0	0	0	0
50	250	260	270	260	22	18.4	21.9	21.4	10.2	10.3	10.4	10.3	30	30	35	0	0	1	1	0
51	300	240	250	310	21.2	22.3	21.4	21.3	10.7	10.8	10.8	10.8	30	0	70	40	0	0	0	0

Master Chart 2 Contd.

S.	Straining				Incontinence				Urgency				Dysuria				Overall Symptoms				Needed Catheterization			
No	B	1m	3m	6m	B	1m	3m	6m	B	1m	3m	6m	B	1m	3m	6m	B	1m	3m	6m	B	1m	3m	6m
27	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
28	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
29	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
30	0	1	0	0	0	1	0	0	0	1	0	0	0	0	0	0	0	1	0	0	0	0	0	0
31	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
32	0	1	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0
33	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
34	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
35	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0
36	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
37	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
38	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
39	0	1	1	0	0	1	1	0	0	0	0	0	0	0	0	0	0	1	1	0	0	1	0	0
40	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
41	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
42	0	1	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0
43	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
44	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
45	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
46	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
47	0	1	1	0	0	1	1	0	0	1	1	0	0	0	0	0	0	1	1	0	0	1	0	0
48	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0
49	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
50	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
51	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0

PATIENT PROFORMA

Name: **Age:** **CD No:**

Height: cm **Weight: kg** **BMI:**

Diagnosis:

Stage (FIGO/AJCC / TNM 7th edition):

Preoperative therapy - Chemotherapy / Radiotherapy / None

Intraoperative Findings:

Postoperative complications:

Final Histopathology:

Postoperative therapy - Chemotherapy / Radiotherapy / None

Voiding symptoms:

Symptoms	Baseline	1st month	3rd month	6th month
Urgency				
Straining				
Urinary incontinence				
Dysuria				
Overall symptoms				
Needed catheterization				

Uroflowmetry:

Parameters	Baseline	1st month	3rd month	6th month
Voided volume (ml)				
Max. flow rate (ml/s)				
Avg. flow rate (ml/s)				
Post-void residual urine(ml)				
Urowflow curve				